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THE

AMERICAN JOURNAL

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OCTOBER, 1915.

ORIGINAL ARTICLES

OUR EXPERIENCES WITH GASTROENTEROSTOMY: A STUDY OF ONE HUNDRED CASES AS COMPARED WITH A SIMILAR NUMBER OF CASES OF PYLOROPLASTY.[1]

By J. M. T. Finney, M.D.,
Professor of Clinical Surgery, Johns Hopkins University,
and
Julius Friedenwald, M.D.,
Professor of Gastroenterology, College of Physicians and Surgeons, Baltimore.

A year ago we reported our thirteen years' experience with pyloroplasty, and from a study of 100 cases the following conclusions were drawn:

1. The operation has certain advantages over gastroenterostomy, and but few of its disadvantages.

2. The operation has its greatest indication in the relief of pyloric stenosis due to chronic ulcers, situated at or near the pylorus, and on either side of it, or resulting from cicatricial contraction following the healing of such ulcers. It is often a useful procedure in cases of hemorrhage due to gastric ulcers on the lesser curvature, or to duodenal ulcers which cannot be controlled medically, and which threaten the life of the patient, as well as in the chronic dyspepsias due to ulcers which have not been relieved by medical treatment.

3. The special advantages of this operative procedure lie in its affording the opportunity to excise all ulcers whether perforated or

[1] Read at the Meeting of the American Gastroenterological Association, in Baltimore, Maryland, May 11, 1915.

not in the anterior walls of the stomach or duodenum after direct inspection of the part affected; also the application of treatment to ulcers situated in the posterior walls. It does not greatly disturb the normal relation between the stomach and intestines, as is the case in other operations.

4. Such objections as are urged against the operation, *e. g.*, its inapplicability in the presence of adhesions surrounding the pylorus, as well as in the presence of active and bleeding ulcers, and also because of the fact that the new opening is not at its lowest point, taking advantage of gravity, are according to our experience more fanciful than real, since the operation has frequently been performed under these conditions with most gratifying results. The interesting experimental work of Cannon and Blake and others supports this contention.

5. The only contra-indications to the operation are inability to mobilize the duodenum when adhesions are too dense, and thickening and infiltration about the pylorus due to hypertrophic forms of ulceration. These conditions, however, in our experience occur but rarely.

6. In atony or gastroptosis with slight motor insufficiency such as is observed in the water-trap stomach, or in nervous dyspepsia, *i. e.*, in gastric disturbances not dependent upon organic disease, this operation is contra-indicated.

7. From our experience with the operation the immediate as well as the final results are most encouraging. While in some instances partial gastrectomy or gastroenterostomy is undoubtedly the operation of choice, nevertheless, on account of its simplicity and because of its satisfactory end results, we believe that pyloroplasty will continue to retain its position as a safe and useful procedure.

Inasmuch as our results with pyloroplasty appeared to be so much more satisfactory than with gastroenterostomy, it seemed to us that it would be well to bring together the results of the later operation, compare them in detail with those of pyloroplasty, and determine whether any advantage really existed in favor of pyloroplasty. For this reason we have collected together 100 cases taken consecutively, beginning in 1902 the date at which the operation of pyloroplasty was first introduced. In gathering our experiences we deem it most important, as with our pyloroplasty cases, that not only should the immediate effects be noted, but also the final results, that is, the results obtained after a term of years.

It is also of importance to note that only those cases have been included in this report in which the gastroenterostomy was performed for other than malignant conditions. All cases in which there has been the slightest question of malignancy have been excluded from this series.

Of the gastroenterostomy cases the immediate results were noted in all cases; the remote results in 74 cases.

136 C 1 20 My 44

In analyzing our results we note that the largest proportion of cases in which it was performed occurred between the thirtieth and sixtieth years.

CASES ARRANGED ACCORDING TO AGE.

Years.	No.	Per cent.	Years.	No.	Per cent.
20 to 25	6	6	45 to 50	12	12
25 to 30	7	7	50 to 55	15	15
30 to 35	10	10	55 to 60	12	12
35 to 40	14	14	60 to 65	5	5
40 to 45	16	16	65 to 70	3	3

Of the total number there are 56 males and 44 females. The following tables illustrate the number of cases observed in males and females according to age:

	Males.		Females.		Total.	
Years.	No.	Per cent.	No.	Per cent.	No.	Per cent.
20 to 25	2	2	4	4	6	6
25 to 30	4	4	3	3	7	7
30 to 35	6	6	4	4	10	10
35 to 40	6	6	8	8	14	14
40 to 45	10	10	6	6	16	16
45 to 50	8	8	4	4	12	12
50 to 55	8	8	7	7	15	15
55 to 60	6	6	6	6	12	12
60 to 65	4	4	1	1	5	5
65 to 70	2	2	1	1	3	3
Total	56	56	44	47	100	100

The duration of the symptoms in our cases varied greatly, ranging from one month to nineteen years, the average duration being nine and a half years.

The following table presents the cases grouped according to the duration of symptoms:

CASES GROUPED ACCORDING TO DURATION OF SYMPTOMS.

Time.	No. of cases.	Time.	No. of cases.
1 month	1	7 years	8
2 "	2	8 "	6
3 "	1	9 "	2
4 "	1	10 "	2
6 "	4	11 "	3
7 "	2	12 "	1
9 "	1	13 "	1
11 "	3	14 "	2
1 year	10	15 "	3
2 years	8	16 "	2
3 "	14	18 "	2
4 "	7	19 "	2
5 "	12		

Average duration of symptoms, nine and a half years.

We have noted the principal symptoms observed in both males and females at various ages. Of these pain was observed in 90 per cent.

of cases (51 males and 39 females); vomiting occurred in 65 per cent. of cases (36 males and 29 females); hematemesis in 23 per cent. (18 males and 5 females); melena in 44 per cent. (28 males and 16 females); retention in 36 per cent. (19 males and 17 females).

PRINCIPAL SYMPTOMS OBSERVED.

Age.	Pain.			Vomiting.			Hematemesis.			Melena.			Retention.		
	M.	F.	Total.	M.	F.	Total.	M.	F.	Total.	M.	F.	Total.	M.	F.	Total.
20 to 30 years	4	6	10	4	5	9	1	0	1	3	6	9	2	2	4
30 to 40 "	11	10	21	8	6	14	4	2	6	7	5	12	6	6	12
40 to 50 "	17	10	27	12	8	20	8	2	10	9	2	11	7	6	13
50 to 60 "	13	12	25	8	9	17	4	1	5	6	3	9	2	3	5
60 to 70 "	6	1	7	4	1	5	1	0	1	3	0	3	2	0	2
Total	51	39	90	36	29	65	18	5	23	28	10	44	19	17	36

In the following table we have arranged the cases of gastric and duodenal ulcers according to age and sex. There are 46 cases of gastric ulcer (32 males and 14 females) and 38 duodenal ulcers (15 males and 23 females) making a total of 84 cases. In addition gastroenterostomy was performed upon 4 cases for gastroptosis, in 2 for pylorospasm, and 9 for pyloric stenosis, due to adhesions (2 due to cholelithiasis, 7 due to adhesions of undetermined cause, probably old healed ulcers). One case presented both gastric and duodenal ulcers.

GROUPING OF THE GASTRIC AND DUODENAL ULCERS ACCORDING TO AGE AND SEX.

Age.	Males.		Females.	
	Gastric. No.	Duodenal. No.	Gastric. No.	Duodenal. No.
20 to 30	4	1	2	3
30 to 40	6	2	2	8
40 to 50	12	5	3	6
50 to 60	8	4	6	6
60 to 70	2	3	1	0
Total	32	15	14	23

Of the 84 cases, obstruction of the pylorus occurred in 67 instances. The obstruction was due in 44 cases to gastric ulcer, in 14 to duodenal ulcer, and in 9 to adhesions. There were 4 cases in which gastroenterostomy was required as secondary to pyloroplasty.

CASES REQUIRING GASTROENTEROSTOMY AS SECONDARY TO PYLOROPLASTY.

Age.	Sex.	Cases.
58 years	F.	Pyloric obstruction due to adhesions produced by gall-bladder infection eleven years after pyloroplasty.
52 "	M.	Duodenal ulcer formed five years after pyloroplasty.
48 "	M.	Infected sutures causing immediate obstruction at site of pyloroplasty requiring gastroenterostomy after five days.
41 "	M.	Duodenal ulcer after one and one-half years.

In one case there occurred a pyloric obstruction due to adhesions produced by cholecystitis eleven years after the pyloroplasty. In the second, a duodenal ulcer formed four years after the pyloroplasty. In the third infected sutures (linen thread) caused early obstruction at the site of the pyloroplasty, and in the fourth, a duodenal ulcer formed after a year and a half. In all of these cases secondary gastroenterostomies had to be performed, which gave entire relief. In our gastroenterostomy cases secondary operations were necessitated in four instances. In one case there was a vicious circle; an entero-anastomosis was made on the day following the operation, and the patient died two days later. In two instances the gastroenterostomy opening became obstructed by adhesions, after one year and one year and a half respectively, secondary gastroenterostomies were performed.

In a fourth instance a secondary gastroenterostomy was performed after the old gastroenterostomy opening had become obstructed after a period of three years.

CASES REQUIRING SECONDARY GASTROENTEROSTOMIES.

Cases.	Age.	Sex.	Cause.
I	70 years	M.	Vicious circle.
II	24 "	F.	Obstruction from adhesions after one year.
III	35 "	F.	Obstruction from adhesions one and a half years.
IV	53 "	M.	Obstruction from adhesions after three years.

It is only fair to call attention to the fact that some of the earlier of the gastroenterostomies were performed by the method then in vogue—namely, the anterior long-loop anastomosis, which did not yield so satisfactory results as those obtained by the no-loop posterior anastomosis. By far the larger number, however, were done after the latter method.

The following table illustrates the immediate results of gastroenterostomy of the 100 cases, consisting of 56 males and 44 females; the operations proved satisfactory immediately in 45 males and 37 females (82 per cent.) and unsatisfactory in 11 males and 7 females (18 per cent.).

In contrast with the immediate results of pyloroplasty, postoperative nausea and vomiting are not infrequent after gastroenterostomy. There are but few instances in our series in which marked nausea did not appear; vomiting was present in 72 of our 100 cases, while flatulency and distention were not infrequent. When one notes the great freedom from discomfort following immediately after pyloroplasty, there can be no question as to the distinct immediate advantage of this procedure over gastroenterostomy.

It is but fair to state, however, that the nausea and vomiting have been at times induced by improper feeding, produced by overdistention and weighing down of the fundus, and thus tending to produce a temporary closure of the new opening. In a number

of instances this condition was overcome by relieving the overdistention and increasing the tone of the stomach by means of lavage.

Diarrhea after operation appeared in eight instances, mainly, however, after the first week or ten days, and was usually due to errors in diet. Inasmuch as the operation is performed in most instances upon patients affected with pyloric obstruction, and after having had this condition for years, it is easily understood how, on account of the rapid passage of the gastric contents through the stomach, a weakened intestine may be overtaxed and intestinal indigestion and diarrhea be induced. On this account, as well as because the stomach has in most instances temporarily lost its tone; and inasmuch as in some cases active ulcers are still present, we have found it advisable to regulate the diet following immediately upon operation and continuing on for several months according to the following plan, which has been found very satisfactory. On account of the frequent occurrence of gastrointestinal upsets after gastroenterostomy we have, according to our experience, found it more important to follow the following dietetic restrictions than after pyloroplasty.

For two days after operation nourishment is administered only by means of rectal alimentation. Normal salt solution or plain water, after Murphy's method, is begun immediately after operation, with nutrient enemata at intervals of every four hours on the second and third days. On the third day after operation, egg-albumin is given in teaspoonful doses, gradually increased to one-half ounce every two hours on the fourth day and two ounces on the fifth day. On the eighth day any liquid is permissible, and on the eleventh day the patient is given a soft-boiled egg; on the thirteenth, soft diet; on the fifteenth, very restricted light diet; on the sixteenth, a restricted light diet; on the eighteenth day, light solid food. Small quantities of water at a time are allowed by mouth after twenty-four hours.

Diet List following the Operation of Gastroenterostomy. First day, first twelve hours, nothing by mouth; nutrient enemata every four hours alternating with continuous salt solution by Murphy's method.

First day, second twelve hours: water in dram doses.

Second day: increase water gradually up to 1 ounce every two hours.

Third day: water 1 ounce, alternating with albumin, 1 dram.

Gradually increase quantities of each until

Eighth day: any liquid 2 ounces every two hours.

Ninth day: any liquid, 3 ounces every two hours.

Tenth day: any liquid, 4 ounces every two hours (discontinue rectal feeding).

Eleventh day: one soft-boiled egg in addition to any liquid.

Twelfth day: two soft-boiled eggs in addition to any liquid.

Thirteenth day: soft diet.
Fourteenth day: soft diet.
Fifteenth day: very restricted light diet.
Sixteenth day: restricted light diet.
Seventeenth day: restricted light diet.
Eighteenth day: any digestible solid food.

After the eighteenth day the following diet list may be gradually followed, and should be continued for at least four or five months: Soups: any light soup. Meats: any of the easily digestible meats, as brains, sweetbreads, beef, mutton, lamb, or poultry (best minced, and taken either broiled or boiled). Fish: mainly the white variety, mackerel, rock, bass, as well as oysters (boiled or broiled). Eggs: in any form except fried. Vegetables: best taken mashed and strained; the easily digestible forms, as asparagus, spinach, peas, beans, potatoes, carrots. Farinaceous food: any of the cereals; bread to be taken stale. Desserts: any of the light puddings. Fruits: mainly stewed. Fatty food: cream, butter, and olive oil. Drinks: milk, buttermilk, cocoa, carbonated mineral water, and plain water.

The following must be avoided: Rich soups, fried foods, pork, veal, stews, hashes, corned meats, potted meat, twice cooked meat, liver, kidney, duck, goose, sausage, crabs, sardines, lobster, preserved fish, smoked fish, salted fish, salmon, cauliflower, celery, radishes, cabbage, cucumbers, sweet potatoes, tomatoes, beets, corn, salads, bananas, melons, berries, pineapple, hot bread, or cakes, nuts, candies, pies, pastry, preserves, cheese, strong tea, strong coffee, alcoholic stimulants.

IMMEDIATE RESULTS OF OPERATION.

Age.	Males.		Females.	
	Satisfactory.	Unsatisfactory.	Satisfactory.	Unsatisfactory.
20 to 30 years	5	1	5	2
30 to 40 "	8	4	10	2
40 to 50 "	16	2	9	1
50 to 60 "	12	2	11	2
60 to 70 "	4	2	2	0
Total	45	11	37	7

In order to obtain a clear view as to the immediate results of the operation we have compared the gastric secretion in the following two cases of pyloric stenosis due to ulcer, before the operation with that obtained one, two, and three months after operation. The figures given are an average of a number of examinations. The total acidity is first noted, then the amount of free hydrochloric acid, and finally the quantity of contents recovered, which in a measure represents the motor activity of the stomach. It is evident in every case that the total quantity of acid as well as the percentage of free hydrochloric acid, which may be exceedingly high before operation, is gradually reduced to normal, but that this does not occur immediately after operation, but requires about two to three

months before the normal is established, after which it continues to remain stationary. Similarly the large quantity of contents obtained is gradually reduced to normal after operation, while the retention observed at first entirely vanishes. It is, therefore, evident that while the secretory and motor functions of the stomach are at once markedly improved after gastroenterostomy, these functions do not usually become normal immediately, but just as in the pyloroplasty cases several months must elapse before perfectly normal conditions are established.

	Total acidity.	Free HCl.	Quantity of contents: After test meal.	On fasting stomach.
M. J. operation: June 27, 1908				
Before operation:				
May 5, 1908	120	74	280	210
After operation:				
July 24, 1908	82	70	100	0
July 30, 1908	62	62	86	0
Aug. 10, 1908	46	38	54	0
Sept. 2, 1908	42	30	50	0
R. R. operation: June 27, 1909				
Before operation:				
May 11, 1909	105	82	310	230
After operation:				
Aug. 2, 1909	100	72	96	0
Aug. 30, 1909	88	66	55	0
Sept. 15, 1909	44	32	45	0

Deaths following Operations. There were seven deaths following immediately upon operation; these occurred from the third to the thirty-third day. In one instance death was due to pneumonia following a secondary gastroenterostomy for a vicious circle; in a second, death was sudden, due to pulmonary embolism; in a third, it was due to toxemia from an intestinal obstruction; in a fourth to persistent vomiting, due to a vicious circle; in a fifth, to exhaustion from a postoperative diarrhea, and in a sixth and seventh to bronchopneumonia in debilitated individuals.

The following table illustrates the number of deaths following immediately upon operation, with cause of death:

IMMEDIATE RESULTS—DEATHS.

No.	Sex.	Day of death after operation.	Cause of death.
I	M.	3	Pneumonia following a secondary gastroenterostomy for a vicious circle.
II	M.	7	Sudden death due to pulmonary embolism.
III	M.	23	Toxemia due to intestinal obstruction from inflammatory growth.
IV	M.	33	Persistent vomiting due to vicious circle.
V	F.	16	Exhaustion, persistent postoperative diarrhea.
VI	F.	5	Bronchopneumonia following one day after operation in a weakened patient; death on fifth day.
VII	M.	4	Bronchopneumonia in a very weak man.

The results of gastroenterostomy during the first year of the operation are fairly satisfactory. Of the 100 cases operated on, 7 had died immediately following operation, and the results of 16 could not be obtained; the results are therefore tabulated in only 77 cases, consisting of 43 males and 34 females; the results were entirely satisfactory in 34 males and 31 females, that is in 65 instances (84.4 per cent.), and unsatisfactory in 9 males and 3 females, that is in 12 instances (15.6 per cent.).

RESULTS OF OPERATION DURING FIRST YEAR.

Age.	Males.		Females.	
	Satisfactory.	Unsatisfactory.	Satisfactory.	Unsatisfactory.
20 to 30 years	4	1	5	1
30 to 40 "	8	1	9	1
40 to 50 "	12	4	7	0
50 to 60 "	8	1	9	1
60 to 70 "	2	2	1	0
Total	34	9	31	3

Of the total of 100 cases the final results of 19 are unknown while 7 died soon after operation, leaving 74 cases. Of these the results were satisfactory in 32 males and 29 females, that is, 61 (84.2 per cent.) and unsatisfactory in 8 males and 5 females, that is, 13 (15.8 per cent.).

RESULTS AFTER FIRST YEAR OF REMAINING SEVENTY-FOUR CASES.

Age.	Males.		Females.	
	Satisfactory.	Unsatisfactory.	Satisfactory.	Unsatisfactory.
20 to 30 years	3	1	1	1
30 to 40 "	8	2	8	2
40 to 50 "	11	3	7	0
50 to 60 "	8	1	9	1
60 to 70 "	2	1	1	1
Total	32	8	29	5

In the following table are noted the number of cases that have been observed from one to eleven years after operation together with the final results. The average of the satisfactory recoveries is shown to be 84.2 per cent. If we deduct from this number 7 per cent. that those cases already referred to, in which death occurred immediately upon operation, the percentage of favorable results is reduced to 77.2 per cent.

In a series of Roentgen-ray plates made in four cases, all of which had been operated on over a year, it was demonstrated that while in most instances the gastroenterostomy opening was functioning quite normally a portion of the gastric contents continued to empty itself through the pylorus.

End Results in Years after Operation of Gastroenterostomy.

Years.	Total No. of cases.	Satisfactory results.	Unsatisfactory results.	Percentage of satisfactory recoveries.
1	9	8	1	88.8
2	5	5	0	100.0
3	8	7	1	87.5
4	7	6	1	85.7
5	4	3	1	75.0
6	7	5	2	71.4
7	5	5	0	100.0
8	9	9	0	100.0
9	7	6	1	85.7
10	8	6	2	75.0
11	5	3	2	60.0
Average of percentage of satisfactory recoveries				84.2
Deaths				7.0
Favorable results				77.2

Fom our observations of a study of 100 cases of gastroenterostomy we believe we are justified in arriving at the following conclusions:

1. The largest proportion of cases in our series in which gastroenterostomy was performed occurred between the thirtieth and sixtieth years, while the largest proportion of cases in which pyloroplasty was performed occurred between the thirty-fifth and fiftieth years.

2. Of the total number of cases of gastroenterostomy there were 56 males and 44 females, while the total number in the pyloroplasty series numbers 63 males and 37 females.

3. The duration of the symptoms in our gastroenterostomy cases varied from one month to nineteen years, the average duration being nine and a half years, while the duration of symptoms in our pyloroplasty cases varied between one and one half months to twenty-five years, the average duration being nine and a half years.

4. In the gastroenterostomy cases the symptoms appeared in the following proportions: pain 90 per cent. (51 males and 39 females); vomiting in 65 per cent. (36 males and 29 females); hematemesis in 23 per cent. (18 males and 5 females); melena in 44 per cent. (28 males and 16 females); retention in 36 per cent. (19 males and 17 females); while in the pyloroplasty cases pain was present in 92 per cent. of cases (58 males and 34 females); vomiting in 64 per cent. (44 males and 20 females); hematemesis in 21 per cent. (15 males and 6 females); melena in 46 per cent. (29 males and 17 females); retention in 42 per cent. (27 males and 15 females).

5. In the gastroenterostomy series the ulcer cases are arranged as follows: there are 46 cases of gastric ulcer (32 males and 14 females), 38 duodenal ulcers (15 males and 23 females), making a total of 84 cases. In the pyloroplasty cases there were 55 cases of gastric ulcer (36 males and 19 females) and 32 duodenal ulcers (21 males and 11 females, making a total of 87 cases. It will be noted that in both series there is a preponderance of males over females, and of gastric over duodenal ulcers.

6. In 67 of the gastroenterostomy patients operated on for obstruction 44 were due to gastric ulcer, 14 to duodenal ulcer, and 9 to adhesions, while pyloroplasty was performed for obstruction in 64 instances; for gastric ulcer in 51, for duodenal ulcer in 7, and in 6 for adhesions.

7. In our gastroenterostomy cases secondary operations were performed in four instances to correct immediate difficulties while in the pyloroplasty cases secondary operations were required in the same number of instances for a similar purpose.

8. After gastroenterostomy satisfactory results were secured in 82 per cent. of cases, and unsatisfactory in 18 per cent., while pyloroplasty was immediately successful in 90 per cent. cases and unsatisfactory in 10 per cent.

9. There were seven deaths, following immediately upon the operation of gastroenterostomy, while but five deaths followed pyloroplasty.

10. Of the 77 cases of gastroenterostomy followed during the first year of the operation the results were satisfactory in 84.4 per cent. and unsatisfactory in 15.6 per cent., while of the 82 cases of pyloroplasty the results were entirely satisfactory in 93.9 per cent., and unsatisfactory in 6.1 per cent.

11. Of the gastroenterostomy cases numbering 74 followed after the first year of operation, the results were satisfactory in 84.2 per cent., and unsatisfactory in 15.8 per cent., while of the pyloroplasty cases numbering 78, the results were satisfactory in 93.6 per cent., and unsatisfactory in 6.4 per cent.

12. The end results in the gastroenterostomy cases showed a percentage of 77.2 per cent. of satisfactory recoveries, while the pyloroplasty cases showed a percentage of 88.6 per cent.

It is quite evident from our study of 100 gastroenterostomy operations and from a similar number of pyloroplasties that the immediate as well as the final results are clearly in favor of pyloroplasty. The only indications in favor of gastroenterostomy are, as we have already pointed out, in those instances in which there is an inability to mobilize the duodenum when adhesions are too dense, and in those cases in which there is a thickening and infiltration about the pylorus due to hypertrophic ulceration, conditions, however, which in our experience occur but rarely. Again there is no possibility of excising the ulcers when performing gastroenterostomy, as can frequently be accomplished in pyloroplasty, when they are in the anterior wall.

While in some instances gastroenterostomy may be the operation of choice, nevertheless we believe that on account of its comparative unsatisfactory end results, it should be as far as possible limited to the relief of stenosis of the pylorus, due to malignant disease, and that usually in nearly all other conditions pyloroplasty and pylorectomy are safer and more satisfactory procedures.

PROLONGED FASTING IN DIABETES.[1]

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Partial pancreatectomy, with preservation of the pancreatic duct so as to avoid atrophy of the remnant, gives a more satisfactory reproduction of clinical diabetes than is afforded by von Mering and Minkowski's total pancreatectomy or Sandmeyer's method of slow pancreatic atrophy. Simple lowering of the sugar tolerance without diabetes, or diabetes varying in intensity from the mildest to the very severe, can thus be produced at will. As previously pointed out,[2] animals thus prepared constitute valuable test-objects and afford favorable opportunities for research concerning diabetic therapy. In the milder types the measures ordinarily used in human diabetes, namely, restriction of carbohydrate or protein and brief fasting, if necessary, suffice to keep the animals sugar-free and in good condition indefinitely. But in a more severe type these measures do not suffice and the animal quickly passes into a hopeless condition if more radical treatment is delayed. In this type the initial fast must sometimes be measured in weeks rather than in days. The subsequent diet must be such as to keep the animal at a low level of weight and metabolism. Certain influences which increase either weight or metabolism tend to bring back glycosuria in these animals as in human patients. If glycosuria persists in any animal the result is a downward sequence of lowered tolerance, emaciation, weakness, cachexia, and death, with parallel degenerative changes in the islands of Langerhans. If glycosuria is prevented, the animals may remain lively and strong though thin; they may improve somewhat with time, and the islands seem to be spared. This evidence supports the hypothesis that one set of influences overtax and injure the internal function of the pancreas, and the opposite set of influences protect and rest the internal function of the pancreas.

The treatment of diabetes at the Rockefeller Hospital has been based upon these animal experiments. The patients admitted have been forty-four, chosen as the most severe out of a considerable number of applicants, and representing a sufficient variety as respects age, social condition, and other factors. Certain points may here be mentioned concerning (1) the initial fast and (2) the subsequent diet.

[1] Read at the meeting of the Association of American Physicians, Washington, D. C., May, 1915.

[2] Allen, F. M., Studies Concerning Glycosuria and Diabetes, Harvard University Press, Cambridge, Massachusetts.

Fasting has been employed by writers from Naunyn to Guelpa. The same writers describe a numerous class of diabetics who are not made sugar-free, but go on with glycosuria and acidosis and die in coma. Other patients are made sugar-free only after months of strict dietary treatment, including occasional single or repeated fast-days. Present experience indicates that the glycosuria in cases of even the severest type of diabetes may with advantage be cleared up by one initial fast. The necessary duration may sometimes be as long as eight or ten days. In one case of maximum severity in a youth, aged seventeen years, the changes accompanying fasting were studied by Dr. Eugene Du Bois in the respiration calorimeter. He found that at the outset the total metabolism was about 8 per cent. above normal and the patient was excreting all or nearly all of the sugar formed from protein and burning practically no sugar at all. In a nine days fast the glycosuria ceased, the total metabolism fell to about 20 per cent. below normal, and the respiratory quotient showed that the sugar formed from protein was being burned. It will be remembered that respiratory quotients in the most severe cases during the "oat cure" do not show increased combustion of carbohydrate. The above metabolic observations are new and constitute important evidence for the functional element in the etiology of human diabetes, since they show to what extent recovery can take place within a few days when the most complete possible rest is given the weakened metabolic function.

Severe acidosis, which is known to be diminished by the shorter fasts previously employed, is still more reduced by the more prolonged fasting. Dangerously weak and emaciated patients have borne the fasting with apparent benefit, giving the impression that they had been suffering more from intoxication than from lack of nutrition. Alcohol is valuable during fasting as a food which does not produce glycosuria, though its use is not essential. Broadly speaking, freedom from glycosuria seems attainable in all cases of uncomplicated human diabetes before there is danger of death from starvation. In a few cases seen personally or described by others, death from some severe complication has occurred before the urine became sugar-free. In such cases to date the complication has been a severe infection, or some condition strongly tending to increase metabolism or produce nervous or circulatory disturbance. The fasting has not appeared harmful even in these few cases where it has not been successful. One case of incipient gangrene and one dangerous carbuncle cleared up rapidly under fasting, and threatening complications of infectious or any other character are considered an indication for the radical treatment as described. No contraindication has been met, unless it be the appearance of nausea, vomiting, and prostration while fasting. One woman died in such an attack; but there were attacks of this character in her previous history, and it is not

positive that either the attack or the death was due to the fasting. One man began to vomit and feel unwell on the seventh day of fasting. He was fed and the symptoms immediately passed off. After two weeks of restricted diet a second fast easily cleared up the glycosuria. This simple precaution apparently prevents danger even in this rare type of cases, and in other cases there has never been any sign of danger or harm.

Though the initial fast, to clear up glycosuria and other symptoms, is generally a very simple matter, the subsequent diet, to maintain this condition, is sometimes difficult. It is unquestionably true, in accord with Joslin's[3] warning, that when glycosuria is abolished and strength diminished by long fasting, and then glycosuria and acidosis allowed to return through improper diet, the last state of that man may be worse than the first. Herein may be found the causes of failure in past attempts of this sort. After the fasting patient has been completely sugar-free for one or two days, feeding is begun as outlined in former papers,[4] and the tolerance of the patient for carbohydrate, protein, and fat is determined. Just as fasting is continued not for any limited number of days, but as long as necessary for sugar-freedom, so also the diet is governed not by any theoretical standard of protein or calories, but by the amount of each food that can be given in each individual case while keeping the urine clear. Under this program even weak and emaciated patients have been subjected to under-nutrition in both protein and calories for weeks or months continuously, with ultimate benefit. Any trace of glycosuria is the signal for a fast-day with subsequent modification of diet, and routine fast-days are often used as frequently as once a week even in absence of glycosuria. While individual details must be reserved to a later publication, two principles in the management of severe cases may be mentioned, namely, the benefit of keeping the patient permanently below weight, and the advisability of restricting the quantity of fat in the diet.

A slight temporary reduction of weight was practiced in some cases by Naunyn, whose example has been widely followed. This was not carried to the point of abolishing glycosuria and acidosis in the severest cases; and generally the attempt has been to maintain these patients at the highest possible level of weight and nutrition, with the idea of helping them to withstand a wasting disease. The present idea is the opposite to this, namely, that the reduction of weight is in itself beneficial to the diabetic condition and serves to spare the weakened function and increase tolerance. This measure, like the others, is carried to any point that may be neces-

[3] Discussion of paper at meeting of Suffolk County Medical Society in connection with Boston Medical Library, December 2, 1914.

[4] Allen, F. M., Jour. Amer. Med. Assoc., September 12, 1914; Boston Med. and Surg. Jour., February 18, 1915.

sary in any particular instance. Sometimes a slight reduction of weight suffices even for a severe case. Again, a well-nourished patient, easily kept free from glycosuria, was reduced by twenty kilograms merely because of a slight stubborn ketonuria and a persistently high blood-sugar. Most patients are able to regain weight to a greater or less extent, but few severe ones are able to return fully to normal weight. Any increase that is possible without return of symptoms is permitted. Any gain that brings back glycosuria or ketonuria is checked. The experience in human patients thus far runs parallel to that in the animal experiments upon which this suggestion was founded.

In the treatment of diabetes heretofore, fat has been freely given and even forced upon the patient. It has been restricted only in certain cases with high acidosis and danger of coma. The doctrine has been that fat does not affect diabetic glycosuria except in very rare "fat-sensitive" patients; also, that the diabetic must receive the number of calories required by his metabolism, plus the number of calories lost as sugar and acetone bodies in the urine, and fat has been considered the best food for crowding in these calories. Anyone can readily convince himself that, in a suitably severe diabetic who is symptom-free for days or weeks on a fixed diet, the addition of some quantitiy of butter or olive oil to the diet will bring back the glycosuria, ketonuria, and other symptoms immediately or within a short time. The feeding of fat alone does not cause glycosuria, and there is no proof that the sugar is formed from the fat; more probably the sugar excretion results from the stimulating effect of fat upon metabolism. The overtaxing of the patient's metabolism by giving fat beyond the limit of tolerance may be an additional explanation of the failure to keep certain patients free from glycosuria and ketonuria under former methods of treatment.

Concerning any proposed treatment, inquiry is proper whether it is new and whether its results are superior to those of the old methods. The following statements can be made regarding these points.

The principle tentatively suggested that increase of weight or metabolism increases strain upon the internal pancreatic function and reduction of weight or metabolism reduces strain upon the internal pancreatic function, is new, and if it proves valid will be a useful general guide in treatment. The animal experiments likewise are entirely new. The general policy of insisting upon prompt and lasting freedom from glycosuria and acidosis in all cases of diabetes, even the severest, is a new one. In addition, the main features wherein the proposed treatment differs from the previously established methods may be summarized under the following five headings. The first two represent differences merely of degree, in that the proposed treatment is more radical than the old; namely,

first, an initial fast sufficient to clear up glycosuria in any case and then one or two days longer; second, a subsequent diet such as to keep glycosuria and acidosis permanently absent, with as many interspersed fast-days as necessary for this purpose. The third and fourth features represent differences not in degree but in kind, and are diametrically opposed to the prevalent teachings; that is, the third opposes the idea that the diabetic should be kept at the highest possible level of weight and strength, and that gain in weight is synonymous with improvement; and it substitutes for this the plan of keeping most severe diabetics intentionally and permanently at a sufficiently low level of weight and metabolism, in the belief that return of symptoms and downward progress is thus prevented. The fourth feature stands opposed to the doctrines that fat feeding does not appreciably influence diabetic glycosuria, and that calories lost in the urine should be replaced by additional calories in the diet, preferably in the form of fat. It opposes to these the observation that addition of fat to a fixed diet suffices to bring back both glycosuria and ketonuria in most severe diabetics, and the principle that the patient's tolerance for fat and calories should be followed in the same way as the tolerance for carbohydrate and protein. The fifth feature consists merely of routine or incidental matters, which are not without practical importance. Among these may be mentioned (1) the diet such as not to overtax tolerance and yet satisfy the patient sufficiently that he will follow it continuously at home; (2) the absence of any specific craving for carbohydrate such as diabetics are supposed to manifest, and the contradiction of the prevalent idea that most severe diabetics cannot be trusted; (3) the avoidance of the need of alkali for more than a few days, and therewith relief from disturbances due either to acidosis on the one hand or to prolonged large doses of soda on the other; (4) the principle of clearing up the urine quickly and devoting the greater part of the stay in hospital to educating the patient, rather than devoting the greater part of the stay in hospital to clearing up the urine and dismissing the patient shortly thereafter; (5) instruction of the patient in the simple means of controlling his own condition, through his diet, his body weight, and the daily testing of his own urine with Benedict's solution.

The immediate results as observed up to the present have appeared uniformly beneficial. Reports from a number of clinicians experienced in the older methods of treating diabetes agree that these results under the new method are more favorable. Also the relative simplicity of the proposed method, and the fact that it stops glycosuria without running any risk of acidosis, makes it available for a large body of general practitioners who have heretofore not felt safe in withdrawing carbohydrate or attempting to stop glycosuria in cases with any marked ketonuria. Patients also generally accept radical treatment with quick decisive results

more readily than the weeks or months of privation heretofore used in stopping glycosuria, and the quick relief from polyphagia, polydipsia, and other symptoms aids further in securing their coöperation. For these reasons it is believed that the innovations described are of some real value. As respects the remote results and the influence on the ultimate prognosis of severe diabetes, longer experience must decide. A question is here involved whether diabetes is an inherently progressive disease or whether it is the simple weakness of a metabolic function. If it is the former, patients must ultimately go down hill and die, though not in diabetic coma, and the benefit will consist only in lengthening their lives and keeping them more comfortable. If it is the latter, downward progress may be indefinitely prevented by avoiding overstrain of the weak function, just as in animals. Among the patients treated thus far, during a variable number of months in the hospital and at home, spontaneous downward progress has not yet been observed. Whatever the ultimate outcome, two conclusions seem justified by present knowledge: (1) that this treatment removes glycosuria and acidosis more quickly and surely than has been the practice heretofore, and (2) that patients do better when glycosuria and acidosis are removed than when they are allowed to continue.

PRESENT-DAY TREATMENT AND PROGNOSIS IN DIABETES.

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It is no exaggeration to say that the advance in the actual treatment of diabetes mellitus during the twelve months just passed has been greater than in any year since Rollo's time, and it should be emphasized that this improvement in treatment comes not by chance, but as a result of patient scientific experimentation, which has been based upon the work of the foremost investigators in both laboratory and clinic. It follows experiments upon animals, and is an answer to those critics who have claimed that animal experimentation in diabetes had failed to justify itself. At the same time better and simpler technical methods for the study of the blood, urine and respiratory exchange, as well as new knowledge of the metabolism of diabetes, have contributed a share to this improvement. The truthfulness of the foregoing is easily shown. One year ago physicians were generally afraid to teach their patients to test their urines for sugar; today the physician feels himself

to so much greater an extent master of the disease that this is one of the first steps to be taken.

The advantage of maintaining the urine sugar-free has been universally recognized, but all have conceded that this was impossible without danger from acidosis and inanition. Fasting and a low diet have been known, but it is only fair to give Allen the credit of first to see the therapeutic significance of inanition upon a severe case of diabetes, second to prove upon diabetic dogs that prolonged fasting would render them sugar-free, and third to have the courage of his convictions and apply this principle to human diabetes. Thanks to Dr. Frederick M. Allen we no longer nurse diabetics—we treat them!

A review of all my private cases of diabetes, undertaken for this special purpose, has convinced me that prolonged fasting has produced results far and away ahead of any methods which I have hitherto employed. For myself, I consider the impression which I have thus obtained of far more value than any statistics which my records afford, but it is almost my duty to present these as well.

TABLE I.—CASES UNDER OBSERVATION MAY 1, 1914, TO MAY 1, 1915.

Cases.	Total.	Traced.	Dead.		Alive.	
			No.	Per cent.	No.	Per cent.
Total	211	211	31	14.7	180	85.3
Old	75	75	11	14.7	64	85.3
New	136	136	20	14.7	116	85.3
Fasted	55	55	6	10.9	49	89.1
Not fasted						

Old cases are those first seen prior to May 1, 1914; new cases are those first seen after May 1, 1914.

TABLE II.—AGE AT DEATH OF FATAL CASES.

Cases.	Total.	0–10	11–20	21–30	31–40	41–50	51–60	61–70	71–80
Old	11	0	1	0	1	2	2	1	4
New	20	3	2	3	0	3	6	1	2

As a matter of fact, I have seen 211 cases (75 old and 136 new) of diabetes in the twelve months from May, 1914 to May, 1915. All of these have been traced and are available for statistics. (See Tables I and II.) Of these 13 are dead, a mortality of 15 per cent. Fifty-five of the 211 cases were fasted and of these 6 died—a mortality of 11 per cent. In this paper I will discuss the causes of death of fatal cases among the number fasted. Case No. 648 was the first case I fasted, and I simply kept her sugar-free a part of one day in May, and then allowed her gradually to return to her former diet. She died in coma June 16, 1914. Case No. 707 was my second case to undergo fasting, and he never actually

became sugar-free. He at once returned home, broke all dietetic restrictions, and died in forty-six days of coma. I had not then gained sufficient confidence in the treatment to impart it to the patients and to prevail upon them to carry it through to the end. Today there is no need of that, for one patient laughs the other half through the fasting days. Another death was Case No. 855, aged seventy years, onset at fifty-eight years, weight 220, who was first seen by me four days after an operation for appendicitis, during which, unfortunately, a large quantity of ether was employed. Despite fasting, which for the most of the time had been nearly absolute, he never became sugar-free. He died of heart failure, without coma, but probably associated with a necrosis of the cells of the liver and pancreas. Case No. 764 was fasted after leaving my supervision by another physician. The patient broke treatment, exercised unusually, and died promptly in coma. He lived some four hundred miles from Boston, and seven miles from the physician in attendance, and was that physician's first case. Case No. 576 was fasted. He died of recurrence of cancer in the scar of the abdominal incision forty-seven days after the growth had been removed from the bladder by Dr. Chute. Case No. 430, who developed diabetes in June of 1908, at the age of fifty-two years, came under my observation in July, 1911, and was fasted for a day in September, 1914. He returned to Canada and died on March 12, 1915, of diabetic coma. The other 49 cases which have been fasted are alive.

Corroborative evidence of the value of Dr. Allen's ideas is the gratifying fact to me that the last death to occur while the patient was under my personal supervision in a hospital was on August 29, 1914. That patient, Case No. 758, had a blood-pressure of 215, and died of coma. Since that date 48 cases have been treated by me in hospitals without a death.

The study of the respiratory quotient of two groups of fatal cases of diabetes and another group of living cases furnishes evidence which is worthy of mention. These investigations were made at the Nutrition Laboratory of the Carnegie Institution in conjunction with Professor Francis G. Benedict, and are illustrated in Tables III and IV. In the first group of fatal cases with a respiratory quotient of 0.71, the duration of life following the period of observation varied between forty-four and fourteen days. In another group of eight fatal cases with the somewhat higher respiratory quotient of 0.74, the duration of life under similar conditions varied from four hundred and forty-two to seventy days. In comparison with these groups of fatal cases, there is a group of four cases still living. The average respiratory quotient of these four cases was 0.715, but the duration of life had already reached, up to May 1, 1915, from eight hundred and sixty-two to one hundred and thirty-four days. These patients are now (June 1, 1915)

all alive and are in good condition. Furthermore, they have been treated along lines employed by Dr. Allen.

TABLE III.—RESPIRATORY QUOTIENT IN SEVERE DIABETES.

Year.	Cases.	Observers.	R. Q.
1894	1	Weintraud and Laves	0.70
1897	2	Nehring-Schmoll	0.72
1905	2	Magnus-Levy	0.71
1907	1	Mohr	0.72
1908–1911	19	Benedict and Joslin	0.73
1912	8	Rolly	0.74
1912	3	Grafe and Wolf	0.74
1912–1914	7	Benedict and Joslin	0.73

TABLE IV.—RESPIRATORY QUOTIENT IN FATAL AND LIVING CASES OF SEVERE DIABETES COMPARED.

Fatal cases.			Four living cases.		
No. of cases.	Days before coma.	R. Q.	Case number.	Days before May 1, 1915	R. Q.
6	44–14	0.71	552	862	0.72
			765	187	0.73
			786	173	0.71
8	442–70	0.74	806	134	0.70
			4 cases	862–134	0.715

The behavior of the various types of diabetes under fasting and a low diet is not quite uniform. The mild and moderately severe cases have done well and time has been saved in the initial treatment. A good many of the moderately severe cases, who frequently showed sugar, now keep sugar-free easily and this has contributed much to their peace of mind. Both conditions may be attributed to the patients having learned to test their own urines. Children have been particularly favorable subjects, although I cannot say that I have a case under treatment which has exceeded that of Case No. 74, a duration of three years and eleven months, which is my record case for a child with an onset under fifteen years.

Complications of pruritus, carbuncles, and abscesses have cleared up better than formerly, and so did one case with neuritis. A woman, Case No. 812, six months pregnant and with 6.4 per cent. of sugar, under a low diet—not fasting—became sugar-free, was delivered by Cesarean section by Dr. J. C. Hubbard, and both baby and patient are now, eight weeks later, in good condition. Other patients have been operated upon successfully for cancer of the uterus, cancer of the bladder, removal of prostrate, amputation for gangrene and for extensive carbuncles.

The results are divergent with long-standing cases. In one notable case, Case No. 801, of ten years' duration, there is no question but that the improvement has been marked, and now he lives comfortably on a diet of 45 grams carbohydrate or less and attends to his customary occupation. Incidentally, the recovery from a carbuncle which led to his first coming to me for treatment, was unusually prompt. But another case, Case No. 352 of twenty-

one years' duration, who was not doing well, although he has become sugar-free, has a tolerance of only 10 to 15 grams carbohydrate. His weight is maintained after an initial loss of four and one-half pounds, but he is weak. I think that his strength will return, but the blood sugar varies between 0.14 per cent., 0.19 per cent. and 0.27 per cent., although no complicating nephritis is present.[1] Another patient, Case No. 664, has steadily lost weight and although she feels fairly comfortable, is weak. Perhaps I should treat this last patient more rigorously—perhaps the diseased pancreatic function is really becoming exhausted. Sometimes it almost seems as if a patient with diabetes, as well as his friends, felt injured if his span of life was not prolonged further than that of a healthy individual. There is no question but that many of the patients of a severe type are less vigorous for some weeks after the fasting period is over than before it began, when they were excreting sugar. In a number of cases time enough has elapsed to show that strength is regained, but this is not universal Perhaps I look for too quick results. However, it would not be fair to say to an active diabetic doctor, with a rather severe type of diabetes, that if he followed this plan of treatment he could return to a strenuous practice in three or four weeks. Of course he ought not to have a strenuous practice, but all of us know that many such physicians do. On the other hand, it would be perfectly correct to say, if you follow this plan of treatment, there is little doubt but that you will live longer, only you must live at a reduced rate.

There remains the type of disease with severe acidosis, and for these patients this form of treatment is almost universally successful. Table V on page 490 illustrates this point.

Unfortunately, in none of the severe cases has there been any decided gain in tolerance, although there have been deceptive apparent gains.

The advantages of the new treatment are many. It has made attainable the ideals of treatment—namely, a sugar-free and acid-free urine. The standards of the success of treatment are so simple that they are within reach of the patient. At one stroke the patient is delivered from medicines, patent and otherwise, sham kinds of treatment, gluten breads, and in 99 cases out of 100 of alkalis. He can now test all measures for himself. Consider the amount of time and money saved both doctor and patient in urinary tests.

The hospital stay is not particularly shortened, except indirectly by the avoidance of complications, because the patient requires a more thorough education in the diet. It does simplify general hospital treatment, because so soon as a diabetic patient in an open ward shows sugar, it will generally mean that he has broken his diet, for which the automatic penalty is a fast until sugar-free.

[1] This patient has continued to improve, looks well and rides horseback. July, 1915.

TABLE V.—CASE NO. 740. MALE. ONSET IN MAY, 1914, AT TWENTY YEARS AND TEN MONTHS.

Date 1915	Body weight.	Volume of urine.	Reaction.	Total nitrogen.	NH_3-N	Acetone and diacetic acid.	Diacetic acid (Qual.)	Beta-oxybutyric acid.	Dextrose (Grav.)	Diet.					Blood sugar.	Alveolar air CO_2 tension.	CO_2 per kilo per min.	O per kilo per min.	R. Q.	Calories per kilo per 24 h from O_2.
										Carbohydrate	Protein.	Fat.	Alcohol.	Calories.					
	Kilos.	c.c.		gms.	gms.	gms.		gms.	gms.	gms.	gms.	gms.	gms.	gms.	%	m.m.	c.c.	c.c.		
March 27	54.0	3000	Acid	32.70	5.60	..	++++	..	74											
April 14–15 (14¼ hrs.)	52.9	1430	Acid	12.44	2.64	3.11	+	19.99	40	..	..	..	0							
" 15–16	52.2	1540	Acid	16.48	3.16	3.70	+	18.33	39	13	50	70	0	882	0.26	24.1	2.91	4.06	0.72	27.5
" 16–17	51.7	1420	Acid	16.20	3.28	3.09	++	13.41	45	17	55	75	0	963	0.43	29.5	3.02	4.15	0.73	28.2
" 17–18	52.1	900	Acid	9.63	2.44	2.00	+	8.23	16	0	7	0	24	196	0.26	31.4	2.93	4.08	0.72	27.6
" 18–19	51.7	680	Acid	8.10	2.14	1.79	++++	7.85	8	0	6	0	0	24	0.33	31.0	2.84	3.87	0.73	27.0
" 19–20	50.6	580	Slightly acid	6.80	1.73	1.29	+++	5.59	3	0	0	0	24	168	0.29	31.6	2.84	3.76	0.76	25.7
" 21–22	51.5	540	Slightly acid	7.90	1.57	1.18	++	4.40	0	0	5	0	24	188	0.23	32.1	2.71	3.61	0.75	24.6
" 24–25	51.6	570	Acid	9.10	1.57	1.09	+++	3.55	0	5	20	25	18	451	0.31	30.8	2.61	3.54	0.74	24.1
" 27–28	50.9	640	Slightly acid	7.40	1.02	0.91	+	3.10	Trace	0	2	0	15	113	0.31	..	2.73	3.67	0.75	25.0
May 1.	50.4	945	Slightly acid	7.60	0.74	0.83	0	2.64	Trace	0	2	0	15	113	0.28	32.9	2.61	3.44	0.76	23.5

Unquestionably complications will become less frequent, and it is possible that arteriosclerosis will less commonly occur in the long standing cases as a result of the restricted diet.

One of the greatest benefits which will be derived from the new method of treatment is the basis which it will give for new lines of experimental work. The question of the D : N ratio should be soon settled. Already new facts have appeared as a result of the study of the metabolism of these patients, and to some of these attention is now directed.

The investigation of the metabolism of severe cases of diabetes has been continued by Professor Benedict and myself at the Carnegie Laboratory, and 19 more patients have been studied since September 1, 1914. The first notable change which we observed was an upward tendency of the respiratory quotient in these cases following the institution of a fast. This became so marked that we had doubts about the accuracy of the respiration apparatus which we employed. A new apparatus, devised by Dr. Benedict, but not yet described, was also employed, and with this the results were essentially the same. Still more recently we have confirmed our data with the Tissot apparatus and the bed calorimeter. In January we compared the results which Dr. Du Bois obtained with two of Dr. Allen's patients, and noted that in his laboratory a similar tendency had been recorded. Table V illustrates this increasing respiratory quotient and other changes in the metabolism. The patient was Case No. 740, a young man in whom the onset of the disease occurred at the age of twenty years and ten months in 1914. He showed, nineteen days before the first test, 5.6 grams of ammonia nitrogen in twenty-four hours, and 74 grams sugar in the urine upon a diet supposedly containing but a few grams of carbohydrate. In June he was sugar-free and at work.

It will be noted that the respiratory quotient was 0.72 upon the first day of observation, but gradually rose to 0.76 upon the last day. If one calculates the non-protein respiratory quotient, it will be seen that this also rose, and it is obvious that notable quantities of carbohydrate, or some material with a high respiratory quotient, must have been burned following the starvation treatment. This remarkable phenomenon is hard to explain. Has the diabetic patient actually more carbohydrate in the body than is commonly supposed? Could the rising quotient be due to combustion of acid products, of which the respiratory quotients for acetone, diacetic acid and beta-oxybutyric acid are 0.75, 1 and 0.89 respectively? The chart shows that if a diabetic is fasted acidosis disappears, and this is in marked contrast to the behavior of normal men, for they present acidosis upon fasting with no increase in the respiratory quotient as the fasting proceeds.

The metabolism at the beginning of the experiment was 18 per cent. higher than at the end. Coincident with this decrease in the

metabolism was the fall in acidosis. It appears, furthermore, logical to conclude that if the metabolism had been estimated a few days prior to the first experiment when the acidosis was greatest of all, amounting to 5.6 grams ammonia-nitrogen, the metabolism would have been still greater. The normal metabolism for this patient lay probably somewhere between the metabolism first and last recorded, and one would not be far wrong to estimate it at the beginning of the test at 10 per cent. above normal and at the end at 10 per cent. below normal. The significance of the fall in metabolism with the decrease in acidosis is striking.

The chart is also instructive in that the falling metabolism bears no relation whatsoever to the blood sugar, for this was essentially the same throughout. It is interesting that the calories per kilo per twenty-four hours, as calculated from the oxygen inhaled, amounted to approximately 28 to 24 per day, and that the calories given the patient during the latter part of the period were approximately this same figure. Subsequent days have confirmed the observation that this patient, when sugar and acid-free, can hold his weight at a little less than 30 calories per kilogram body weight.

The method which has been employed in the treatment of these cases has been similar to that recommended and described by Dr. Allen. The more closely his directions have been followed the better the patients have done. No patient has required more than five days to become sugar-free. Many patients became sugar-free after the omission of three or four meals. Patients are put to bed and the plan of treatment is carefully explained to them. They are furnished with note-books in which answers to their questions are recorded, taught the use of a diet card and how to examine the urine for sugar with Stanley R. Benedict's solution. As a working basis the general plan of treatment is summarized below, and for convenience the following diet chart, printed on a card five by three inches, has been used.

Fasting. Fast until sugar-free. Drink water freely and one cup tea and one cup coffee if desired. If sugar persists after two days of fasting, add in divided portions 300 c.c. clear meat broth.

Alcohol. If acidosis (diacetic acid) is present, take 0.5 c.c. alcohol per kilogram body weight daily until acidosis disappears. Alcohol is best given in small doses every three hours.

Carbohydrate Tolerance. When the twenty-four hour urine is sugar-free, add 150 grams of 5 per cent. vegetables, and continue to add 5 grams carbohydrate daily up to 20 grams, and then 5 grams every other day, passing successively upward through the 5, 10 and 15 per cent. vegetables, 5 and 10 per cent. fruits, potato and oatmeal to bread, unless sugar appears or the tolerance reaches 3 grams carbohydrate per kilogram body weight.

STRICT DIET. MEATS, FISH, BROTHS, GELATINE, EGGS, BUTTER, OLIVE OIL, COFFEE, TEA AND CRACKED COCOA.

Foods Arranged Approximately According to Per Cent. of Carbohydrates.

5 per cent.	10 per cent.	15 per cent.	20 per cent.
VEGETABLES			
Lettuce	Onions	Green peas	Potatoes
Spinach	Squash	Artichokes	Shell beans
Sauerkraut	Turnip	Parsnips	Baked beans
String beans	Carrots	Canned lima beans	Green corn
Celery	Okra		Boiled rice
Asparagus	Mushrooms		Boiled macaroni
Cucumbers	Beets		
Brussels sprouts			
Sorrel			
Endive			
Dandelions			
Swiss chard			
Sea kale			
Cauliflower			
Tomatoes			
Rhubarb			
Egg plant			
Leeks			
Beet greens			
Water cress			
Cabbage			
Radishes			
Pumpkin			
Kohl-rabi			
Broccoli			
Vegetable marrow			
FRUITS			
Ripe olives (20 per cent. fat)	Lemons	Apples	Plums
Grape fruit	Oranges	Pears	Bananas
	Cranberries	Apricots	
	Strawberries	Blueberries	
	Blackberries	Cherries	
	Gooseberries	Currants	
	Peaches	Raspberries	
	Pineapple	Huckleberries	
	Watermelon		
NUTS			
Butternuts	Brazil nuts	Almonds	Peanuts
Pignolias	Black walnuts	Walnuts (English)	
	Hickory	Beechnuts	40 per cent.
	Pecans	Pistachios	Chestnuts
	Filberts	Pinenuts	

MISCELLANEOUS

Unsweetened and unspiced pickles, clams, oysters, scallops, liver, fish roe

Reckon *actually available* carbohydrates in vegetables of 5 per cent. group as 3 per cent., of 10 per cent. group as 6 per cent.

Contain approximately.	Protein.	Fat.	Carbohydrates grams.	Calories.
Oatmeal, dry weight	5	2	20	110
Meat (uncooked)	6	2	0	40
Meat (cooked)	8	3	0	60
Broth	0.7	0	0	3
Potato	1	0	6	30
Bacon (cooked)	5	15	0	155
Cream, 40 per cent.	1	12	1	120
Cream, 20 per cent.	1	6	1	60
Milk	1	1	2	20
Bread	3	0	18	90
Butter	0	25	0	240
Egg (one)	6	5	0	75
Brasil nuts	5	20	2	210
Orange (one)	0	0	10	40
Grape fruit (one)	0	0	10	40
Vegetables, 5–10 per cent. groups	0.5	0	1	6

1 gram protein, 4 calories.
1 gram fat, 9 calories.
6.25 grams protein contain 1 gram nitrogen.
30 grams (g) or cubic centimeters (c.c.) = 1 ounce.
A patient "at rest" requires 25 to 30 calories per kilogram body weight.

1 gram carbohydrate, 4 calories.
1 gram alcohol, 7 calories.
1 kilogram = 2.2 pounds.

PROTEIN TOLERANCE. When the urine has been sugar-free for two days, add 20 grams protein (three eggs) and thereafter 15 grams protein daily in the form of meat until the patient is receiving 1 gram protein per kilogram body weight, or if the car-

bohydrate tolerance is zero, only ¾ gram per kilogram body weight. Later, if desired, the protein may be raised to 1.5 gram per kilogram body weight.

Fat Tolerance. While testing the protein tolerance, a small quantity of fat is included in the eggs and meat given. Add no more fat until the protein reaches 1 gram per kilogram (unless the protein tolerance is below this figure), but then add 25 grams fat daily until the patient ceases to lose weight or receives not over 40 calories per kilogram body weight.

Reappearance of Sugar. The return of sugar demands fasting for twenty-four hours or until sugar-free. The diet preceding the reappearance of sugar is then resumed except that the carbohydrate should not exceed half the former tolerance until the urine has been sugar-free for two weeks, and it should not then be increased more than 5 grams per week.

Weekly Fast Days. Whenever the tolerance is less than 20 grams carbohydrate, fasting should be practised one day in seven; when the tolerance is between 20 and 50 grams carbohydrate, 5 per cent. vegetables and one-half the usual quantity of protein and fat are allowed upon the fast day; when the tolerance is between 50 and 100 grams carbohydrate, the 10 per cent. and 15 per cent. vegetables are added as well. If the tolerance is more than 100 grams carbohydrate, upon the weekly fast day the carbohydrate should be halved.

Bread is seldom prescribed, because it is so easy for a patient to overstep the limits. Many patients use bread substitutes, such as Huntley & Palmer's Akoll Biscuits, Barker's Gluten Flour[2] (Brand A), Hepco Flour,[3] Lyster Bros'. Diabetic Flour, Whitefield, New Hampshire. The quantity of fat which it is necessary to give a severe case is considerable. A diabetic weighing 60 kilograms requires at least 30 calories per kilogram body weight to be up and about the hospital, with an occasional walk. Since in the severe cases not more than 10 grams carbohydrate, representing 40 calories, can be given in this form, and seldom more than 75 grams protein (1.25 grams per kilogram body weight) which would amount to 300 calories more, the balance of the diet must be made up of 150 grams fat, amounting to 1350 calories, and even more unless 15 grams alcohol are given, which would amount to 105 calories.

Table VII.—Quantity of Food Required by a Severe Diabetic Patient Weighing 60 Kilograms.

Food.	Quantity grams.	Calories per gram.		Total calories.
Carbohydrate	10	4	=	40
Protein	75	4	=	300
Fat	150	9	=	1350
Alcohol	15	7	=	105
		Total	=	1795

[2] Herman Barker, 433 Broadway, Somerville.
[3] Waukesha Health Products Co., Waukesha, Wisconsin.

The quantity of fat (150 grams to 160 grams) is easily given in the form of 120 c.c. of 40 per cent. cream (48 grams), 15 grams of oil (15 grams) and 3 eggs (15 grams), bacon (cooked) 30 grams (15 grams), meat, cooked, 100 grams (5 grams), and butter 60 grams (50 grams). These figures are only approximately correct.

Should the patient remain sugar-free and the weight be maintained upon this diet, gradually the quantity of fat could be lowered and the carbohydrate increased. A very few of my patients have a tolerance for between 200 and 300 grams of carbohydrate. With most, the tolerance is below 100 grams, and with the majority it is under 50 grams.

The patient should have one day of restricted diet each week, no matter how mild the case. This is done partly to spare the function which controls the carbohydrate metabolism, but also to remind the patient of what a strict diet really is. The patient is told to gain little or no weight, and as Dr. Allen advises, not to come up to his former weight. The severer cases examine the urine daily, and the milder ones once a week. The patients are instructed to lead less strenuous lives. Unfortunately, they feel so well that often this advice is disregarded, and I believe that all of us err in allowing our patients to do too much. They should have nine hours in bed at night, and should have a quiet hour of rest each day, no matter how well they feel.

Patients must be followed up. The secret by which this is brought about lies in instructing the patient how to live in the first few days of treatment, and making him understand that he must always be under supervision. This is sometimes difficult, for a doctor does not like to force a patient to come to him. Fortunately, improvement in the treatment of diabetes is rapid enough to enable a physician to repay his patient for a visit with new ideas at comparatively short intervals.

The after treatment of diabetic patients is quite as important as the initial treatment. Patients must learn to keep sugar-free and maintain weight, and when difficulties occur report for advice. There is not the slightest doubt, in my opinion, that even under the old regime the patients who were kept under judicious supervision did better than those who neglected treatment.

Many dangers suggested themselves when I commenced to use the fasting treatment, but most of these have gradually faded away. It is still true that a patient who undertakes the treatment and then relapses to former slack methods is worse off than his untreated brother. Liability to infection is not common. A child, Case No. 813, of eight years acquired tonsillitis and developed an otitis media, but this was a circumstance not unusual for any hospital patient. In another direction an unlooked for danger has appeared. Owing to the apparently easy control of diabetes, several diabetic married women have seen no reason why they should not become pregnant. So far I have treated but one case under the new regime,

and that patient did well. How other patients will do I cannot predict.

More difficulties will arise in the introduction of the newer methods of treatment from the physician than from the patient. Physicians who have only an occasional case of diabetes under their care, and in general mild ones, will not take the time or see the necessity of keeping their patients sugar-free. It is the same experience with which we are all familiar in pulmonary tuberculosis. A number of physicians who have had serious cases have become interested and investigated the treatment, and these report excellent results. In several instances fasting has been carried out most successfully by the family physician at home. The difficulty will arise in prevailing upon the physician to carry out protracted fast in the severer types of the disease. This takes real courage, and it is desirable that all who have had good results with severe cases should publish their reports. The doctor in the hospital clinic can easily handle the patient, but it takes time to educate the family and all the friends.

No one can tell how well diabetic patients will do in the future. It is surprising to me to note how much better my patients have done during the last six months, and to note that those cases which have followed most carefully the restricted diet and fasting have done the best. So far, no patient whom I have taught to do his own urine during the last year has died. I am coming to feel that coma no longer represents the culmination of the disease, but that it is an avoidable accident.

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RELATIVE PULMONARY INSUFFICIENCY (GRAHAM STEELL MURMUR).

By Joseph Sailer, M.D.,
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In 1888 Graham Steell,[1] in discussing the physical signs of mitral obstruction and regurgitation, wrote as follows: "Mitral diastolic

[1] Med. Chron., 1888, viii, 89.

murmurs of less intensity are, however, not infrequently heard best to the left of the sternum just below the pulmonic area about the sternal end of the fourth rib and downwards for a little way over the right ventricle. Again, a still finer, in fact a soft blowing diastolic murmur, is not very rarely to be heard in the pulmonic area itself as well as for a short distance below it. There can be no question of the occurrence of such a murmur in a few cases of mitral stenosis, but its interpretation has been a matter of dispute. It is difficult to accept the view that it is a direct mitral murmur owing to the softness, and it has been suggested that it is really a murmur of coexisting aortic regurgitation. The diastolic murmur of aortic regurgitation may undoubtedly be propagated into this region, as if by predilection, when it is feeble; but in the cases of mitral stenosis in which I have observed this murmur there was no evidence of aortic regurgitation in the pulse or otherwise. I have thought that the murmur in question may be explained by a trifling leakage taking place through the valves of the overdistended pulmonary artery, just as we know that regurgitation occurs under similar conditions in the case of the aorta. I once observed a case of cirrhosis of the lung which I interpreted in this way."

In 1906 he wrote[2] as follows: "The diastolic murmur in the pulmonic artery may, as already noted, accompany congenital stenosis, being due to the coincident deformity and incompetence of the valves. Such a murmur is not a rare occurrence in cases of long-standing mitral stenosis. When first established it usually comes and goes according to the degree of blood-pressure within the vessel. It is accompanied by dilatation of the pulmonic artery, which, as already stated, under the circumstances may become atheromatous like the aorta. This murmur has been called the murmur of high pressure in the pulmonic artery."

It would appear from this that Steell had frequently heard this murmur, but in an article published between the appearance of his first article on the subject of mitral stenosis and his text-book upon *Diseases of the Heart*, he gave a statistical analysis of fifty cases of mitral stenosis. Discussing the diastolic murmurs heard in these cases, he states that in Case 46 there is some ground for belief that the murmur representing the murmur of high pressure in the pulmonic area which he had described was present. It appears, therefore, that this murmur was doubtfully present in not more than 2 per cent. of his cases, and probably, had his observations been extended, in a considerably smaller percentage. Although he speaks of the fact that the occurrence of this murmur is associated with dilatation of the pulmonic artery, he brings forward no definite proof that such a dilatation occurs, for it does not appear in any case that he found such a dilatation postmortem. At least no

[2] Diseases of the Heart, Manchester, 1906, p. 105.

such finding is mentioned, and it seems reasonable to conclude that it would have been.

This murmur has attracted little, if any, attention from other clinicians. It is not mentioned by McKenzie nor by Hirschfelder, nor have I, to the best of my recollection, come across reference to it in literature excepting in the articles by Steell himself. Nevertheless, it seemed to me not unreasonable that such a murmur should occur, and in all cases in which there was reason to suspect high pressure in the pulmonary system I have attempted to discover its existence. I believe that I have been able to identify it in three cases. It is heard as a soft, blowing systolic murmur, smooth, usually rather distinct in character from other murmurs in the heart, heard most distinctly just beneath the pulmonic cartilage, and transmitted obliquely downward and to the right to a point just below the junction of the third costal cartilage and the right border of the sternum. It commences with the second sound and terminates before the first. In none of these three cases in which I think I heard it was it loud. There was no accompanying thrill. The second sound was distinctly audible, but not accentuated. Of these three cases, one died and permission was refused for autopsy, one passed from observation, and the third is the case I wish to report.

W. M., male, white, aged seventy-eight years, was admitted to the Philadelphia Hospital, January 21, 1907, complaining of dyspnea and loss of appetite. He apparently improved and was transferred to the out-wards, where he remained until, on account of increasing disability, he was transferred to my service on the medical floor the latter part of October, 1914.

The family history was negative. He did not recall any of the diseases of childhood. He had had rheumatic fever about twenty years ago, but otherwise had been in good health until his admission to the hospital. He denied syphilis or gonorrhea. At times he drank heavily, chiefly whisky. He did not recall any injuries or surgical operations. For the past three years he had noticed that his wind was poor. There was constant slight dyspnea, increased by any exertion. He had a chronic cough and expectoration, but the sputum was not bloody. Sometimes he suffered from constipation. For three days before his admission to the medical ward he had been vomiting greenish material. This was the first sign of gastric disorder.

The notes of the physical examination are as follows: A well-nourished adult white male; contracted but normally reacting pupils; poor teeth; apex beat in the sixth interspace one-quarter of an inch to the left of the mammary line. The first sound at the apex was distinctly loud. The second pulmonic sound was quite distinct. There was a diastolic murmur heard at the junction of the left third costal cartilage of the sternum which could be traced

downward and to the right for a short distance. It was of a soft, blowing character and terminated before the first sound. The second aortic was distinctly accentuated. The heart's action was irregular. The pulse was small and irregular. The artery was palpable. There was no indication of a Corrigan pulse. There was evidence of consolidation at the apex of the left lung and some rales at the base of both lungs. The liver and spleen were not enlarged. There were no areas of tenderness in any part of the abdomen and no abnormal masses. The fingers were moderately clubbed. There were brown pigmented areas in the tibial regions of both legs.

FIG. 1.—View of mitral valve through the left auricle, showing the extreme stenosis.

A diagnosis was made of hypertrophy of the heart; arteriosclerosis; mitral disease and possibly Graham Steell murmur. In addition, tuberculosis of the left apex was noted as probably present, and it was supposed that there was an old pleuritis of the right base. A Roentgen-ray examination was made and reported a dilated aorta.

The patient grew steadily worse. On October 17 there was Cheyne-Stokes breathing and the vomiting continued. On October 18, a loud presystolic rumble was heard at the apex, and a loud systolic murmur, which was transmitted to the left but not into the

vessels of the neck. The arrhythmia had become very pronounced. On November 3, Cheyne-Stokes breathing had returned and the patient was very weak. The pulse was feeble and arrhythmical. The presystolic rumble had disappeared. Both lungs were full of rales. On November 7 the patient died. The urine was negative. There was no blood count and no Wassermann reaction. A second Roentgen-ray, made particularly with reference to the lungs, reported that they were apparently normal, that there was considerable dilatation of the aorta, and that the heart was moderately hypertrophied, particularly on the right side.

FIG. 2.—View of cavity of the left ventricle, the aortic valve, and the first portion of the aorta. All pictures made with lens at same distance from the heart.

An autopsy was made the following day and the notes are exceedingly minute, and are interesting partly because of the things not observed, partly because of the things found that were not suspected. Of the latter the most important was cholelithiasis, ulcerated cholangeitis, acute fibrous pericholecystitis, and cholangeitis. In spite of the Roentgen-ray, tuberculosis was found at the apex of the left lung, and numerous adhesions were found at the base of the right pleura. A small amount of fluid was found in the pleural cavities. The notes on the heart may be given in full. They are as follows: "The heart shows the left ventricle dilated,

with some thickening of the wall. Musculature fairly firm, cloudy appearance. Endocardium grayish. Aortic leaflets thickened along the base. Papillary muscles show fibrous tips. Pulmonary leaflets show congenital fenestrum. Coronary arteries show several elevated thickenings along course." . . . "Spreads made from curtained material in gall-bladder show encapsulated diplococci, probably pneumococci and an unidentified bacillus."

The specimen was preserved at my request, and upon examination the following notes were made: The wall of the left auricle is

FIG. 3.—Partial view of the cavity of the right ventricle of the pulmonary valves and the first portion of the pulmonary artery.

greatly thickened. There is a buttonhole mitral valve, the orifice of which is irregular, its greatest diameter being less than five millimeters. The wall of the left ventricle is only moderately hypertrophied. The aortic leaflets are slightly thickened. The aortic orifice is 4.6 cm. in circumference. The wall of the right ventricle is slightly thickened. The circumference of the pulmonary orifice is 7.8 cm. The circumference of the pulmonary artery, 2 cm. above the orifice, is 7.8 cm., and of the aortic artery, 2 cm. above the orifice, is 4.7 cm. The pathologist's notes may therefore be amended to read "hypertrophy and dilatation of the left auricle;

extreme mitral stenosis; dilatation of the pulmonary artery and moderate dilatation and hypertrophy of the right ventricle."

It is, I think, of some interest that the Roentgen-ray examination was at fault in two particulars: the dilatation was of the pulmonary artery and not of the aortic, and the lungs were not normal. The pathologist is apparently at fault in overlooking the mitral stenosis and the dilatation of the pulmonic artery. As the mitral stenosis was not recorded either in the autopsy notes or in the pathologist's diagnosis, it is fair to assume that the mitral valve was not particularly examined. If the heart had not been preserved it would of necessity have been assumed that mitral disease did not exist, and that therefore the clinical observation of a Graham Steell murmur was inaccurate. At the subsequent examination, however, the existence of mitral stenosis and of dilatation of the pulmonary artery in conjunction with the physical signs observed during the patient's life accord with the conditions postulated by Graham Steell as necessary for the occurrence of the murmur, and it seems reasonable to conclude that a functional insufficiency of the pulmonary valve secondary to the mitral stenosis was present in this case.

The value of the discovery of the Graham Steell murmur is not at the present time evident. Unquestionably in those cases in which pressure on the pulmonary circulation is so increased that the artery is dilated and the pulmonary valves thereby become incompetent the prognosis is not so favorable, but it does not appear in these cases that restoration of compensation is therefore impossible. It may be that with the accumulation of observations more definite significance can be attached to it. It does indicate, however, quite definitely and positively in all the cases in which I have seen it, the existence of decompensation, and hitherto always of a severe type.

THE SOY BEAN AND CONDENSED MILK IN INFANT FEEDING.

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In 1909 I called attention to the soy bean,[1] and suggested its use in infant feeding. Two years later, having become convinced of its value, I submitted a report, with a short article,[2] before the American Pediatric Society. In the meantime, in order to bring the subject before the medical profession, another article was pub-

[1] The Soy Bean in Infant Feeding, Preliminary Report, Arch. Pediat., July, 1909.
[2] Further Observations on the Soy Bean, Ibid., October, 1911.

lished.[3] In addition to its use in infant feeding the bean has been used in diabetes, and Friedenwald and myself called attention to the work which had been done on this subject, and published[4] an account of our own experience in this regard. This article brought so many requests for information about the preparation of the bean, that a short article on its cooking followed.[5]

Inasmuch as the bean is still practically unknown in this country, it may not be out of place to review briefly the facts in connection with it. The soy bean (Glycine hispida), sometimes incorrectly called the soja bean, is an annual leguminous plant, which originally grew in a wild state from Cochin China to the south of Japan and to Java. It has been cultivated since the very earliest times in Japan, and in China long before the time of Confucius. The bean gradually found its way into India, but has been very slow in spreading to other lands. Something over one hundred years ago it was brought to Europe and grown in botanical gardens chiefly as a curiosity. It was described by numerous writers, among the very early contributions being one by Koempfer, who observed it in his travels throughout the East, and wrote about it in 1712 under the name of Daidsu. Linnaeus described it under the name of Dolicha soja, and since then many others have written about it, notably Montigny, one of the French consuls in China in 1854, and following his suggestion, in 1855, there was an attempt made to introduce it into France under the auspices of the Société d'acclimatation. It was introduced into England about 1790, but there do not seem to have been any studies made upon it from the economic standpoint. In 1875 Haberlandt started a series of investigations in Austria-Hungary, and became an enthusiastic advocate of it, publishing a book about its use as food for both man and animals. As early as 1829 Thomas Nuttall wrote an article concerning the bean as a valuable crop for the United States. When the Perry expedition returned from Japan, in 1853, the beans were again brought to this country, but up until the last fifteen or twenty years the plant seems to have been known only as a curiosity. Recently, however, studies have been made in the various agricultural experiment stations, most notably those of Kansas and Massachusetts, and since 1898 the Office of Seed and Plant Introduction of the United States Department of Agriculture has taken up the work of distributing the bean. In this country it has been grown chiefly as a forage crop, and very little attention has been paid to its use as food for man.

Like many other leguminous plants it absorbs nitrogen from the atmosphere, and is frequently used for enriching the soil, the entire

[3] The Soy Bean as an Article of Diet for Infants, Jour. Amer. Med. Assoc., 1910, liv, 1664.

[4] The Use of the Soy Bean as a Food in Diabetes, Amer. Jour. Med. Sci., 1910, cxl, 793.

[5] Soy Bean Cookery, Med. Rec., September 23, 1911.

plant being plowed under after it is grown upon more or less barren land. The plant itself is an erect annual, with branching stems covered with short hairs. The leaves are trifoliate and more or less hairy, and the flowers are violet or pale lilac in color. The pods contain from two to five seeds, and the different varieties of the bean vary in size, shape, color, and in the length of time they take to mature. Some are whitish, some yellow, some greenish-yellow, some brown, and some black. In shape some are spherical and some are more or less compressed. The plant varies from two to four feet in height, and bears a remarkable number of beans. The flowers are self-pollinated, which makes the yield independent of insects. Ordinarily the plant grows best in the same temperature as corn, but the different varieties differ in the length of time they take to mature, and the more rapid varieties may be grown in the North. Seven different varieties are handled by seedsmen, and some twenty-two distinct varieties are known, while in China and Japan almost every village is said to have its own strain of soy beans. The bean should be harvested early, as the pods open spontaneously, and if allowed to stay on the plants, scatter the beans on the ground. The ordinary variety ripens in from seventy-five to ninety days. A full account of the plant will be found in the *Farmers' Bulletin*, No. 58, of the United States Department of Agriculture, and of the different varieties in the *Bureau of Plant Industry*, Bulletin No. 98.

The bean has been put to many uses. It is grown for silage and as a hay crop, and is used to improve the quality of the soil. The bean has been used as food for animals, especially cows and pigs. Some varieties contain large quantities of oil, which is used as a food and for illuminating purposes, as a lubricant, and also for the manufacture of paint and soap. The residue left after the oil has been expressed may be used as a fertilizer and also as food for animals.

The beans are eaten as a vegetable, in soups, sometimes picked green, boiled, and served cold with a sprinkling of soy sauce, and sometimes served as a salad. The bean forms the basis of the so-called soy sauces used as condiments all over the world. In the East it is most frequently used in the form of more or less cheese-like foods that are prepared from it. Of these there are a great variety, the best known being natto, tofu, miso, yuba, and shoyu. Natto is a sort of bean cheese made by boiling the beans until they become very soft, and then placing the resulting mass in a warm cellar, where it ferments. Tofu is prepared by soaking the beans for about twelve hours in water, and then crushing them between mill stones. They are then boiled in about three times their bulk of water and filtered. The filtrate is a white, opaque, milky liquid with a taste similar to malt. In the making of tofu the protein is precipitated by adding the mother liquid obtained by the manufacture of salt from sea water, the precipitate being made into cakes. The chemical analysis of the filtrate is as follows:

	Per cent.
Water	92.53
Protein	2.02
Fat	2.13
Fiber	0.03
Ash	0.41
Nitrogen-free extract, including carbohydrate	1.88

The large amount of nitrogen which these beans contain probably account for the small amount of meat and other animal food taken by the Japanese. According to the dietary studies made in Japan the nitrogen from the beans, especially if taken in a mixed dietary, is easily absorbed.

The soy beans are sometimes roasted and then used as a substitute for coffee. They are also sometimes served as a vegetable, first soaking the beans until the skins come off, and then boiling them until soft and serving hot. The North Carolina Experiment Station recommends the bean as a palatable vegetable, and suggests that the bean be soaked until the skins come off and they be stirred until the skins rise to the surface, when it is easy to remove them. The beans are then boiled with bacon until soft, seasoned with pepper, salt, and butter, and served hot. If the beans are green the preliminary soaking may be omitted.

In my first experiments I used the beans, but for purposes of making gruel for infant feeding they require a considerable amount of cooking, so at my request Mr. Deming, of the Cereo Company, of Tappan, New York, made a flour which is entirely satisfactory for practical use. An analysis of this flour yielded the following results:

	Per cent.
Protein N. X. 6.25	44.64
Fat	19.43
Mineral matter	4.20
Moisture	5.26
Crude fiber	2.35
Cane sugar	9.34
Non-nitrogenous extract	14.78
Starch	None
Reducing sugars	None
Polarization normal weight due to optically active substance other than cane sugar included in proteins and non-nitrogenous extract	7.86°

The percentage of protein in this flour is almost one-third greater than the percentage of protein in the whole beans. This is caused by removing the coarse, fibrous hulls which contain little protein.

Each ounce contains 13 grams protein and 120 calories.

	Protean, per cent.	Fat, per cent.	Sugar, per cent.	Calories.
¼ ounce, 1 level tablespoonful to quart	0.35	0.15	0.08	30
½ ounce, 2 level tablespoonfuls to quart	0.70	0.30	0.15	60
¾ ounce, 3 level tablespoonfuls to quart	1.00	0.45	0.23	90
1 ounce to quart	1.40	0.60	0.30	120
2 ounces to quart	2.80	1.20	0.60	240

Condensed milk as a food for infants has, of course, been well known for many years. In most instances it has been used only as a temporary expedient, either when pure milk cannot be obtained, or in traveling, or for infants who have been improperly fed on mixtures containing too much fat and protein. Condensed milk has the following compositions:

Condensed milk (Eagle Brand):

	Per cent.
Fat	9.82
Milk sugar	12.49
Protein	8.80
Ash	1.90
Cane sugar	40.50
Water	26.49

This furnishes about 102 calories per ounce, and in dilutions with water, as ordinarily used, it contains the following percentages:

	1 in 6, rarely used.	1 in 8.	1 in 12.	1 in 16.
Fat	1.66	1.25	0.83	0.62
Protein	1.50	1.12	0.75	0.56
Sugar	8.83	6.63	4.41	3.31
Calories per ounce approximate	17.00	12.8	8.50	6.40

After a considerable amount of clinical experience I found that the best method to begin feeding mixtures of soy bean and condensed milk is to make a gruel composed of one level tablespoonful of soy flour, two level tablespoonfuls of barley flour, a pinch of salt, and one quart of water. This should be boiled for twenty minutes, or even longer, and the water lost in the cooking should be replaced. To this is added condensed milk, varying from 1 to 16 to 1 to 8, according to the age of the child and other circumstances. As a rule this will be found to agree admirably, and may be used in quantities varying from 2 to 8 ounces at a feeding. The feeding interval may be two, two and a half, or three hours, usually three hours, and occasionally, in difficult cases, this may be lengthened to three and a half or even four hours; but I have not found this advisable in many cases. The amounts of condensed milk and the strength of the gruel may be varied according to circumstances, using weaker mixtures if there is vomiting, or if there are symptoms of indigestion, or if the stools, after a number of days, do not appear to be more or less normal. The stools of babies fed with soy bean mixtures are slightly brownish in color, should be perfectly well digested and smooth, not unlike in appearance those of babies that have been fed upon malted milk. In children who are not gaining, and yet who seem to be taking the gruel well, the amount of condensed milk may be increased to 1 to 8, that is, 1 ounce of condensed milk, with enough gruel to make 8 ounces, and in exceptional cases, even to 1 to 6, though I have rarely found this either advisable or necessary. The gruels may vary in composition, and in older children

they may be increased to double the strength advised above, that is, 2 level tablespoonfuls of soy flour and 4 of barley, and occasionally this may be increased still further. If the soy bean is used too strong, or if it is used without a sufficient amount of carbohydrate with it, as in perfectly plain gruels without either cereal flours or condensed milk, it is liable to cause thin, dark-colored, foul-smelling stools, owing to the high protein and fat content, and the very small amount of carbohydrate present.

These mixtures of condensed milk and soy bean gruels will be found one of the most valuable additions to the dietary of the infant. On two occasions I have encountered a curious idiosyncrasy for barley flour in these mixtures. Following administration of the gruel made with barley flour, there was a sudden and marked general edema, lasting several days. It so happened that I had seen this condition from barley gruel used alone, so that this was stopped in both instances, and the gruel subsequently made with wheat flour in place of the barley flour. In both instances a second attempt to replace the wheat flour with barley was followed by the same edema, so that there can be little question as to the cause of it. As in feeding any cooked food to infants, some antiscorbutic food should be given, and for this purpose orange juice in quantities from a teaspoonful to two tablespoonfuls may be used daily, or at intervals or several days, or even once a week. I have never seen a case of scurvy resulting from these mixtures, but in all cases in which the gruel was used over long periods of time, some antiscorbutic food has been added.

The class of cases and circumstances in which this mixture is to be advised may be outlined as follows: When fresh milk cannot be obtained, or when the milk supply is very questionable; in summer, when there is some question concerning the milk supply and then in instances where the infant is found to be incapable of digesting cow's milk. It will also be found useful in certain cases of intestinal indigestion in infants who, under ordinary circumstances, can digest cow's milk, but who have had their intestinal functions temporarily deranged. It may also be of service in cases of chronic vomiting, and particularly useful as a food after summer diarrhea. Without the condensed milk the gruel may be used as a food in many diarrheal conditions with considerable advantage, but as is the case with every other food, there are many instances in which it will not agree with the child. The use of soy bean gruels alone without barley or some other cereal may be recommended under very exceptional circumstances. The failure to obtain proper results from the use of the soy bean has been largely due to an attempt to use it without the addition of some cereal. In the past six years I have had a sufficient amount of experience with this method of feeding to know that it can be used without any danger. It will not produce rickets, and with a very small amount

of some antiscorbutic food, will not produce scurvy. It permits a perfectly normal development as far as bones are concerned, and in successful feeding the infants present the appearance of breast-fed babies. In some of the most difficult cases children are slow to reach the normal appearance, but most of such children that I have had have been cases which I do not believe could have been raised by any other means or only by using a wet nurse.

In order that this may be made more plain a few brief clinical histories will be given.

CASE I.—W. L. C., born June 17, 1910. Weighed eight pounds at birth. Was nursed five weeks and then weaned, owing to the failure of the mother's milk. Since that time has been fed on various mixtures of milk, using Walker-Gordon milk and various diluents. It was subsequently put upon various proprietary foods, and also upon 1 to 8 condensed milk mixtures. It was first seen October 9, 1910. The weight at this time was nearly ten pounds. The baby slept well at night, and the stools were normal, rather scanty. It was exceedingly irritable during the day, and it vomited most of its food. The mother stated that the vomiting was about the same on everything that had been tried. Various milk mixtures were tried in rather rapid succession for the next month, with no result, and the weight varied between nine and ten pounds. The baby at times would be more comfortable, but was decidedly doing badly. Patient was put upon the standard gruel suggested above, with condensed milk. Gain in weight began almost immediately, and on December 4 its weight was 11 pounds 4 ounces, and the baby was returned to a milk mixture, on which it did fairly well, and by January 20, 1911, reached a weight of 14 pounds 4 ounces. At this time the food was very badly borne, and in the next two weeks the weight had dropped to 13 pounds 15 ounces, and the vomiting was again very troublesome. It was again put upon the condensed milk and soy bean mixture, and this time was kept upon this until October. The child gained steadily, and on May 25 weighed 20 pounds. This child had rather an uneventful history subsequently, except that until about a year ago was not able to digest cow's milk even if diluted. Since that time it has lost this peculiarity, and is able to digest cow's milk, although it is only with difficulty that it can be induced to take it.

CASE. II.—O. S., one of the best examples of the value of this form of feeding that I have seen. Born April 21, 1914. Second child, first having died at birth; weight, 8 pounds. Was seen April 27, when it had dropped to 7 pounds 4 ounces, was crying continuously, had loose, greenish stools, appeared to have some severe toxemia, slightly jaundiced. The baby continued to nurse, but seemed extremely ill, vomited a great deal, abdomen was extended with gas to an extent that I have rarely ever seen, and there seemed to be considerable difficulty in getting relief from this condition.

Various means were tried, including bismuth and chalk mixtures, betanaphthol bismuth, lactic acid bacillus tablets, and aromatics. The child cried day and night, was unable to pass the gas either by mouth or by bowel, had numerous small greenish stools some days, and at other times was very constipated, when bowels only moved with difficulty. Owing to the worry and care of the child the mother's milk failed, and the child was first put on condensed milk, subsequently upon various modifications of milk from the Walker-Gordon laboratory. These mixtures were started at 2.5 fat, 6 sugar, 0.75 whey protein, 0.25 casein, 5 per cent. lime water and water to make 20 ounces for the twenty-four hours. The feeding time was varied from two hours to three and three and a half, but the condition remained unchanged. During the next month the usual variations were made. Changes in the percentages of fat, sugar, and protein were all unavailing. The child remained practically stationary in weight, sometimes losing a couple of ounces, sometimes gaining a couple of ounces. A second trial was made with the bulgara tablets, and subsequently milk modified with sodium citrate was tried. On July 1 the child had a remarkable attack of edema, lasting four days. This came on suddenly and was very marked, presenting the appearance of a bad case of acute nephritis; but the urine was normal, and the general condition of the child did not seem to be much disturbed. The child was then tried on half mother's milk, obtained from a wet-nurse, and half modified milk, and on July 12 a wet-nurse was obtained. Her milk was normal and the baby took it readily, but the condition remained unchanged. Owing to difficulties with the wet-nurse, this had to be discontinued. The child was taken to the country, where it came under the care of another physician. The first part of this period it was put to a wet-nurse again, but without success.

On July 21 it was put on Loeflund's Malt Soup Extract, 1 tablespoonful, flour 2 tablespoonfuls, milk 6 ounces, and water 18 ounces. Eight bottles, 2 ounces each, were fed every two and a half hours. This was increased to $2\frac{1}{2}$ ounces on July 22 and 3 ounces on July 23. Stools were well digested, but there was considerable difficulty with gas. The Malt Soup Extract was increased slightly, and on July 24, the baby had a severe diarrhea—twelve stools a day. This continued until July 27, when there was still considerable mucus and some blood in the stools. Baby was given Mellin's Food, one ounce in 8 ounces of water, 3 ounces every three hours, to alternate with three breast feedings.

July 30. Mellin's Food, 1 ounce, milk 4 ounces, and water 7 ounces. Four ounces were given at a feeding. The weight during all this period was approximately 7 pounds 9 ounces. Some days it gained an ounce or two, some days it lost an ounce or two.

On August 7 it was given peptonized milk 6 ounces, Mellin's Food 1 ounce, and water 14 ounces; four bottles of 4 ounces each,

and 3 breast feedings. On this it increased on August 10 to 7 pounds 12 ounces, and on August 12 to 8 pounds 1 ounce. It was then given Mellin's Food 2 tablespoonsful, cream ½ ounce, peptonized milk 8 ounces, water 12 ounces; 4 ounces at a feeding, subsequently increased to 4½. On this mixture the baby was distinctly better, the abdominal distention improved, the stools thin, but apparently well-digested.

August 21. Weight 8 pounds 4 ounces.

August 28. Given peptonized milk, 9 ounces, water 11 ounces, Mellin's Food 3 tablespoonfuls.

August 31. Weight 8 pounds 14 ounces. Then followed an attack of indigestion. The baby was again placed on breast milk. There being no improvement on this, it was discontinued.

September 7. Mellin's Food 1½ ounces, milk 6 ounces, water 14 ounces; 4½ ounces at feeding, 7 bottles. Weight 8 pounds 7 ounces.

September 10. Mellin's Food 3 tablespoonfuls, milk 8 ounces, water to make 35 ounces. There was marked abdominal distention, and the stools varied, some being well digested, others containing curds, and being greenish in color. From this time up until October 23, patient was given various mixtures, sometimes malted soup, sometimes peptonized milk, and various formulæ.

The baby returned to town, and again came under my care on October 23. At this time it was taking a mixture of malted soup and flour and milk and water; was in almost constant pain; the abdomen was enormously distended. It was placed on standard soy bean gruel, using wheat flour in place of barley, and condensed milk 1 to 8, 4½ ounces to a feeding, The weights on this gruel were as follows:

November 10. Weight 10 pounds.

November 17. Weight 10½ pounds.

December 3. Weight 10 pounds 13 ounces; feedings increased to 5 ounces.

December 8. Weight 11 pounds 9 ounces.

December 15. Weight 11 pounds 13 ounces; feedings increased to 6 ounces. Stools on this mixture have been normal; baby still has a great deal of gas, but is much more comfortable. Had one severe attack of bronchitis.

December 23. Weight 11 pounds 15 ounces.

December 27. The baby was greatly disturbed from some unknown cause, acute diarrhea.

December 29. Weight 11 pounds 12 ounces; diarrhea stopped.

January 4, 1915. The baby was coughing a great deal, with a great deal of wheezing.

January 6. Weight 12 pounds 7 ounces.

January 13. Weight 12 pounds 13 ounces.

January 20. Weight 13 pounds 7 ounces.

January 27. Weight 14 pounds 2 ounces

February 3. Weight 14 pounds 5 ounces.
February 10. Weight 14 pounds 10 ounces.
February 17. Weight 15 pounds 2 ounces.
March 25. Weight 17 pounds 9 ounces.

Only a few of the difficulties of this case are noted above, but the account gives a fair idea of what was encountered, omitting numerous incidents which did not have any effect upon the child's development.

CASE III.—W. J. S., born September 28, 1913; first child, normal labor. Weighed at birth 6½ pounds. First seen on May 23, 1914. This baby was nursed for four weeks, was then put on modified milk, using a large number of different formulæ, none of which agreed with the child. The baby gained a few ounces from time to time, but remained very thin, and presented a typical marantic appearance, vomited almost continuously, and cried all the time. Following the use of modified milk was a period in which condensed milk, malted milk, and various modifications with Mellin's Food, were used. In February the child was removed to an infant hospital, and eventually placed on breast milk, and after three months of this treatment the baby was returned home, having gained up to eleven pounds. While in the hospital he was changed to a mixture of milk and water, and was given three level tablespoonfuls of cooked farina three times a day. During the month prior to my seeing the baby he had gained two ounces. The child was exceedingly uncomfortable. Nothing abnormal was noted in the physical examination beyond what was stated above. An effort was made to increase the milk, and an addition was made of barley water to it; but this was not very successful, although the baby gained five ounces on it, it continued to be very uncomfortable and to vomit.

On June 1 the baby was changed to a mixture of soy bean and barley, one level tablespoonful of the former, and two of the latter to one quart of water. To this was added condensed milk 6 drams, with sufficient gruel to make six ounces. Six feedings of this were given at three-hour intervals. By June 13 the baby weighed 11 pounds 9 ounces; by July 6, 12 pounds 2 ounces.

On August 1 it weighed 15 pounds and became upset from overfeeding. During this period I was out of the city.

On September 14 it weighed 14 pounds 5 ounces, and at this time he had been on a mixture of cow's milk and oatmeal water.

On September 26 milk and egg were added every other day to the food, and subsequently the gain in weight was rather uneventful.

On October 31 it weighed 16 pounds 3 ounces, and on February 15, 1915, it weighed 22 pounds. During this time he had been on a mixture of 40 ounces of milk, 2 ounces of lime water, and 8 ounces of diluent, part of the time barley water, part of the time oatmeal water, and part of the time plain water. In addition to this he was given farina and other cereals, with the addition of the soy

bean flour cooked with the cereal. The appearance of the child is now practically normal.

CASE IV.—L. C. F., born January 1, 1913; first child, normal labor. Weighed 8 pounds 12 ounces at birth, and gained steadily until March 30, when it weighed 11 pounds 2 ounces, and continued to lose weight until April 5, when it weighed 10 pounds 15 ounces. The mother's milk was found to be low in fat and rather scanty, and the usual means of improving the milk were unavailing. The child was weaned and placed upon a mixture of milk, barley water, and milk sugar, which agreed very well, but on which the child did not gain in weight.

On May 2 the child was given standard mixture noted above of soy bean, barley water, and condensed milk, and from this time on the baby averaged about 4 ounces a week gain, and a perfectly uneventful year following, presented the appearance of a breast-fed baby. During this year the place of residence was frequently changed, and the baby was kept upon the mixture to prevent the child getting spoiled milk, in many of the places there being no adequate milk supply.

CASE V.—J. M., born July 11, 1913. Child weighed 6 pounds 14 ounces at birth, was nursed up to about October 25. Highest weight reached was 8 pounds 12 ounces about the end of September. It was first seen November 13, 1913, when it weighed 8 pounds 7 ounces. It was on a mixture of milk and oatmeal water. Was vomiting some and gradually losing weight. Was placed upon the standard soy bean and barley gruel, started on 2 drams condensed milk, with sufficient gruel to make 4 ounces, and fed at three-hour intervals. It was gradually increased up to 1 to 8.

On November 17 the baby weighed 8 pounds 12 ounces, and presented a perfectly normal and comfortable appearance.

On December 13 weight 11 pounds, and from this time the patient had a perfectly uneventful year. At the end of the first year it weighed 22 pounds, perfectly normal in every way, and had no digestive disturbances.

A STUDY OF SPLENIC ENLARGEMENT IN EARLY SYPHILIS.

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WHEN one considers the uniformity with which the entire lymphatic system is attacked early in the course of acquired syphilis,

it might naturally be expected that the spleen as well as the other lymphatic structures would take part in this general hyperplasia. It is a surprising fact, however, that an exceedingly scant literature exists which has to do with hypertrophy of the spleen in the early stages of the infection. The numerous texts on syphilology make but brief mention of the fact, and all seem agreed that it is only occasionally that one finds enlargement of the spleen in acute syphilis.

From the results of our studies, it seems beyond a peradventure of a doubt that these brief and cursory references in the literature on enlargement of the spleen in early syphilis are not due to its non-occurrence, but rather to the fact that the subject has received from clinicians but very little study. This is probably due to the fact that syphilis is, and has been for the most part, an ambulatory disease. Hospitalization of the syphilitic is observed only in a few institutions, and a careful study of the visceropathies incident to secondary syphilis are, for this reason, not always possible.

The first reported case of splenic enlargement in association with secondary syphilis was published in 1862 by Biermer.[1] This author noted in a woman, aged twenty-eight years, eight months postinfectionem, marked enlargement of the liver and spleen, together with a roseola. Under treatment the spleen rapidly decreased in size, the liver, however, remaining enlarged.

The credit for first calling attention to the acute splenic hypertrophy in secondary syphilis belongs to A. Weil.[2] This author, in 1874, published observations on three cases of secondary syphilis in which he was able to percuss enlargement of two and palpate a definite tumor in the third. Mention must also be made of the observations of Gold,[3] who, in 1880, published the report of a postmortem on a case of secondary syphilis in which slight enlargement of the spleen was noted. Inasmuch as this patient also had an erysipelas, it may be questioned whether the splenic enlargement was due to this cause rather than to the syphilis.

In 1895 Columbini[4] stated on the basis of his investigation that enlargement of the spleen was a uniform finding in all cases of fresh syphilis, coincident with the appearance of the exanthem. Discussing Columbini's results, Bruhns[5] in 1899, called attention to the fact that the former's results were largely the result of percussion of the enlarged organs, at the same time pointing out the great difficulties in determining splenic enlargement in this way.

This last author investigated 60 cases of recent syphilis, and was able to reëxamine them frequently during the entire first months

[1] Schweizerische Ztschr. f. Heilk., 1862, i, 118.
[2] Deutsch. Arch. f. klin. Med., 1874, xiii, 317.
[3] Vierteljahrsschrift f. Derm. u. Syph., 1880, xii, 463.
[4] Instituto dermosifilopatico della universita di Siena, 1895, cited by Bruhns.
[5] Deutsch. Arch. f. klin. Med., 1899, lxiv, 450.

of treatment. In addition to these sixty cases of early syphilis he examined four of precocious malignant syphilis. The spleens were examined twice weekly and percussion of the organ was always supplemented by palpation. It is surprising to note that of these 60 early cases, Bruhns was able to demonstrate definite enlargement in only 4. In the precocious malignant cases 2 out of the 4 were found to have enlarged spleens. Bruhns found, therefore, 10 per cent. of his early cases with definite palpable tumors, and in not one of these was there any other possible explanation for the enlargement considered present. In 5 out of the 6 cases the enlarged spleen rapidly disappeared under the influence of mercurial therapy.

The next observation of note was that of Wewer,[6] in 1876. This author found enlargement of the spleen 6 times in 79 cases of secondary syphilis. Haslund,[7] who has perhaps devoted more space than any other author, either before or since his time, to splenic syphilis in general, expressed himself as believing splenic tumor in early syphilis as a rare finding.

Nolte[8] examined 50 cases of secondary syphilis and found enlargement in only 2. The largest figures next to those of Columbini are those of Avanzini,[9] who found enlargement in 8 out of 30 cases, slightly more than 25 per cent. Schuchter,[10] in 1887, reported 6 cases out of 22 in which the spleen was enlarged; figures almost identical with those of Avanzini, who, however, found only 16 cases of enlargement of the spleen in 490 cases examined. Unfortunately, however, it is not noted how many of these 490 cases were recent infections, and Bruhns calls attention to the fact that many were probably in the latent stage and later period of the disease.

The great difference of the figures quoted is undoubtedly due to the differences in the methods of examination. It need hardly be mentioned here that percussion of the spleen in determining slight deviations from the normal in its size is wholly unsatisfactory. The figures, therefore, which are based upon this means of physical examination can hardly be reliable. Not only may the difference in the percussion note be modified by the position of the stomach or colon, but, on the other hand, splenic dulness may in some cases be impossible to elicit under the most favorable circumstances. On the other hand it is very difficult to explain the large number of spleens found by palpation alone, by us, as compared with the small number reported by some of the earlier observers.

During the past two years at the University Hospital, observa-

[6] Deutsch. Arch. f. klin. Med., 1876, xvii, 459.

[7] Haslund, Arch. f. Derm. u. Syph., 1882, xiv, 346; Hospitalstid, 1882, Nos. 2 and 3.

[8] Inaug. Dissert., Greifswald, 1883.

[9] Vierteljahrsschrift f. Derm. u. Syph., 1884, xvi, 379.

[10] Wien. med. Bl., 1887, Nos. 41 and 42, pp. 1290 and 1320.

tions have been made under ideal conditions. At our institution syphilis is not treated in the ambulatory fashion. The average length of the time that patients remain is about eighteen days; a large part of this time is spent in bed, and careful observations on such patients are therefore made possible. We have selected for the purpose of this study 100 cases of early syphilis; of these 9 cases were primary syphilis alone, the remaining 91 were cases in the first months of the infection, for the most part with cutaneous or mucous membrane involvement.

METHODS OF STUDY. The patients were studied with particular reference to several different factors. Thus in each case there were noted the general adenopathy; blood findings; the general health; type of eruption; involvement of the bones; involvement of the nervous system; the course of the splenic tumor, if this were present.

GENERAL HEALTH. Under the heading of general health is meant the constitutional disturbances attributable to the infection; slight anemia with a loss from two to five pounds in weight; mild asthenia was rated as a slight effect on the general health. A moderate effect on the general health was regarded as including a blood picture of 3,500,000 to 4,000,000 red cells, a hemoglobin of 70 per cent., a sharp drop in weight from 5 to 15 pounds, malaise, anorexia, and in some cases bone involvement. Under severe involvement of general health are included those cases in which there was marked prostration, loss in weight from 15 to 50 pounds (syphilitic cachexia), severe central nervous disturbances, and cases with pronounced anemias.

It must also be noted that very few of our cases had received treatment prior to their entry into the hospital. In all cases careful examinations were made to exclude the possibility of other infections as possibly causing the splenic tumor. Realizing the inaccuracy of percussion, we have included in this study only those cases in which the spleen was definitely palpable.

GENERAL SUMMARY OF THE FINDINGS. Of the 100 cases examined, definite splenic enlargement was found in 36. The spleen was noted as being hard and firm in 17 cases, in 6 it was noted as tender, and in 3 as very soft.

Of the 36 positive cases the general health was definitely affected in 27 and the remaining 9 showed no impairment of the general functions. Severe involvement of the general health was found in 4 cases. This entailed in 1 case a loss of 50 pounds in weight. Expressed in terms of percentage, 78 per cent. of those cases in which the spleen was enlarged were cases in which the general health suffered considerable deterioration. On the other hand it must be noted that the general health was affected in 22 per cent. of the cases in which enlargement of the spleen was not present.

Definite involvement of bone, including under this heading

ostalgia, arthralgia, periostitis, and arthritis, was found in 15 of our 36 cases, or in 41 per cent.

Central Nervous System. Of the 36 cases in which the spleen was found to be enlarged, 25 were available for the study of the spinal fluid, although all were subjected to a careful neurological examination. Of the 25 cases in which both the neurological examination and the spinal fluid was studied, 19 showed definite involvement of the nervous system. Such involvement took the form of basal meningitis; meningomyeloencephalitis; neuroretinitis; loss of bone conduction, as shown by the functional test of the cochlear and vestibular portions of the eighth nerve; sluggishness of the pupillary reactions; seventh nerve palsy. Of this last rare finding there were 2 cases in our series. Expressed in terms of percentages, therefore, in 76 per cent. of those cases in which the spleen was enlarged there was definite involvement of the nervous system.

Type of the Exanthem. Studying the figures here it is interesting to note that of the 36 positive findings, 19 occurred coincident with a papular efflorescence; 16 cases of the 36 had associated mucous membrane findings, 4 had alopecia. In 2 out of our 9 primary cases the spleen was definitely palpable; in both of these there was as yet no general adenitis, nor was there any sign of hematogenous infection.

Blood Examination. The blood examinations in this series were for the most part done by the senior students of the University of Michigan, and as each blood count was not controlled by us, we feel that too much stress should not be placed on these figures. It is interesting to note, however, that in 27 cases in which counts were made (presumably carefully), 14 showed a leukocytosis of over 10,000, and of 40 cases in which blood examinations were made and in which no splenic tumors were demonstrable, 16 had a leukocytosis of over 10,000. Expressed briefly then there was a preponderance of leukocytosis in those cases in which the spleen was enlarged.

With regard to the type of the splenic enlargement, by far the largest number were firm and readily palpable. The average enlargement extended about two finger-breadths below the costal margin. One cannot describe this type of hypertrophy any more accurately than by saying it closely resembles in consistence the spleen of chronic malaria. The softer spleens, of which there were 19 in our series, presented fully rounded margins, and were distinctly of the type that one encounters in typhoid fever and other acute infections.

We were particularly interested in noting the course of the splenic tumor under proper antisyphilitic therapy. In general, we must conclude from our study that the enlargement disappears much more slowly than do the other earlier manifestations. This

observation parallels that with regard to the slow disappearance of the general adenitis. In one of our cases there was a persistence for over three months, and in a second the spleen resumed its normal size only after eight salvarsan injections were given. In one case the enlargement disappeared after a single injection of salvarsan. The largest number persisted until from three to five injections had been given and until mercury had been begun. The most interesting finding appeared in one case in which prior to the injection of salvarsan the spleen was not palpable, but became so following the first injection and became larger following the second injection of salvarsan.

The incidence of pain and tenderness on pressure were noted in 6 cases, in one at least pain was noted as exquisite. In this case, however, although the enlargement persisted for a considerable time, the pain was very promptly relieved by the antisyphilitic treatment.

We are not in a position to state definitely as to the time of the appearance of the splenic enlargement. In all but one of our cases, as has been noted, the enlargement was found on the first examination. Inasmuch as we found enlargement, however, in two cases of primary syphilis in which indeed the secondary manifestations never developed, it seems reasonable to assume that this condition may antedate the appearance of the other secondary manifestations.

The question naturally arises as to which of the types of splenic enlargement, hard or soft, develop into the later, or interstitial splenitis of late syphilis. With regard to this we noted in general that the softer spleen receded much more rapidly than did those of the firmer consistency. It seems not unlikely, therefore, that the harder persistent types are those which one might expect to develop the later interstitial splenitis, the softer tumors resembling more those of an acute infection and receding with the disappearance of the infectious agent from the blood.

Inasmuch as we based our findings solely on palpation, it must be very evident that 36 per cent. does not represent the total number of splenic enlargement in the cases examined, but simply the number in which the magnitude of the spleen had reached such size that it could be felt below the costal margin. We are inclined to believe, therefore, with Columbini and with Avanzini that the spleen is enlarged in a great majority of all cases of syphilis which go untreated during the first months of infection.

The interesting feature in connection with the persistence of the splenic enlargement lies in the possibility that this organ acts as a reservoir for the spirochetes or their "rest forms," and that the subsequent tertiary manifestations may possibly be due to reinfection of the blood stream from the spleen rather than, as is generally accepted now, that such lesions are recurrences *in loco* of early dissemination. Analogy at least is possible in the cases of

malaria in which the spleen acts as the main point of redistribution of the organisms.

From the results of our studies we venture the following conclusions:

1. Acute splenic enlargement occurs in a large number of cases of early syphilis.
2. This enlargement may be present before the secondary manifestations are apparent, and probably represents the earliest syphilitic visceropathy.
3. The enlargement is either in the form of a soft tumor not unlike that of any other infection or may present itself as a firm, tense tumor.
4. In a small proportion of cases, tenderness, occasionally extreme, may be present.
5. The enlargement is most frequently found in those cases in which the general health has been seriously involved.
6. Acute splenic enlargement in early syphilis tends to disappear under treatment, but may persist and resist treatment longer than any other early manifestation.
7. A striking finding was the frequency with which the splenic enlargement was noted in association with early central nervous manifestations.
8. In no cases in our series was the spleen tumor associated with enlargement of the liver.

CHRONIC LEAD-POISONING IN GUINEA-PIGS: WITH SPECIAL REFERENCE TO NEPHRITIS, CIRRHOSIS, AND POLYSEROSITIS.*

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SAN FRANCISCO.

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The experiments recorded in this paper were undertaken primarily with the object of repeating Charcot and Gombault's[7] work on experimental chronic lead nephritis in guinea-pigs. The results of our experiments along these lines are somewhat disappointing, but in the course of the experiments certain interesting changes were obtained in the livers and the serous membranes of the animals experimented upon, which seem to justify more extensive publication.

* Short notes on this work have appeared in the Proceedings of the Society for Experimental Biology and Medicine, 1912, x, 49–52.

The experiments were performed exclusively upon guinea-pigs, 28 in all. The substance introduced was the same in all cases, carbonate of lead, and it was used in the same way in all experiments, being given by mouth with the food of the animals. As a rule, about ½ grain was given three times a week, but the amount given varied somewhat in different experiments. Since the object of the experiments was to keep the animals under the influence of lead for as long a period as possible, the administration was stopped whenever untoward symptoms developed; that is, whenever the animals began to suffer seriously in their nutrition or when they developed convulsions, which were very commonly observed. On the other hand, many of the animals were given as much lead as they would tolerate, in order to get as definite results as possible.

Since the animals frequently refused their food when lead was added to it, and as they often spilled much of it in the taking, the amount of lead received by each animal cannot be given to a certainty.

All animals eventually died as a result of the continued intoxication, although many of them, except for occasional convulsions, continued in good health and in a good state of nutrition to the last. In fact, some put on an excessive amount of fat, which occurrence in similar experiments has been commented on by other investigators, for instance, by Paviot.[18]

The blood in all cases showed the typical lead anemia. Stippled cells and nucleated erythrocytes were often plentiful in the circulation. Among the latter, megaloblasts were observed frequently. At autopsy the spleen was usually somewhat enlarged; in a few cases considerably so. The venous sinuses were distended and full of erythrophages. The splenic pulp, the marrow, and the liver contained hematogenous pigment in varying amounts. In the marrow the nucleated red cells were extremely numerous and the number of megaloblasts was sometimes very large, more especially so in the long-continued experiments.

The experiments were continued for a much longer time than in any previous series of similar experiments, 13 out of 28 animals surviving for one year or more, 8 of these for two years and more, and 3 even for three years and more. One animal lived almost four years.

The Kidneys. Lead evidently affects the epithelium of the kidneys in a characteristic manner. Its action resembles that of other epithelial poisons, like uranium, chronium, etc., except that it is of a much milder character. The ill effects are almost entirely localized in the distal parts of the convoluted tubules, which have been mistaken for portions of the loops of Henle by some observers. As shown in Fig. 1, the change consists in a granular degeneration of the protoplasm and a pyknotic change in the nuclei. It terminates in necrosis of the cells affected. In few cases the necro-

sis was preceded by fatty degeneration. Eventually the protoplasm either disintegrates, breaking up into little fragments (Fig. 1), or the cells become detached from the wall of the uriniferous tubules

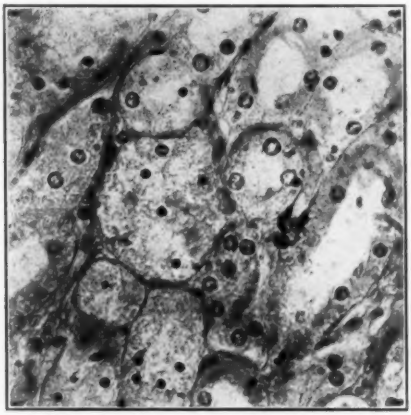

FIG. 1 (× 350).—Granular degeneration of epithelial cells in distal parts of convoluted tubules associated with pyknosis of nuclei and terminating in necrosis. Experiment 4. Guinea-pig 03.

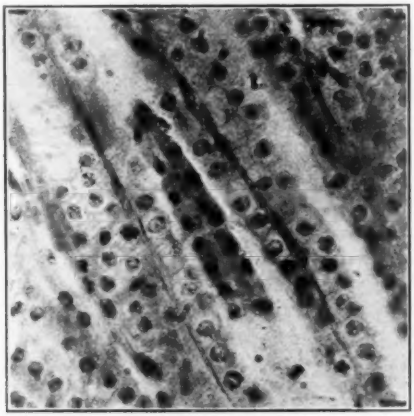

FIG. 2 (× 350).—Epithelial cast in lower part of uriniferous tubule. Experiment 18. Guinea-pig 51.

in toto. In this latter case large epithelial casts may be found in the lower parts of the tubules (Fig. 2). In some cases these are plentiful. Hyaline casts were observed only occasionally. This

process seems to continue to practically the same extent throughout the course of the experiments, the destroyed epithelium being constantly regenerated. Mitoses may be found in fair numbers in all specimens, and in the later stages large atypical epithelial cells with giant nuclei (Fig. 3) are often encountered. Regenerated and degenerated cells are frequently seen in the same section of the same tubule. The rest of the tubules are slightly involved, if at all, except in a few cases, where the lesion is more diffuse and involves the more proximal parts of the convoluted tubules.

Examination of the urine at autopsy or during the life of the animals sometimes revealed a slight trace of albumin and commonly degenerated renal epithelium, rarely casts.

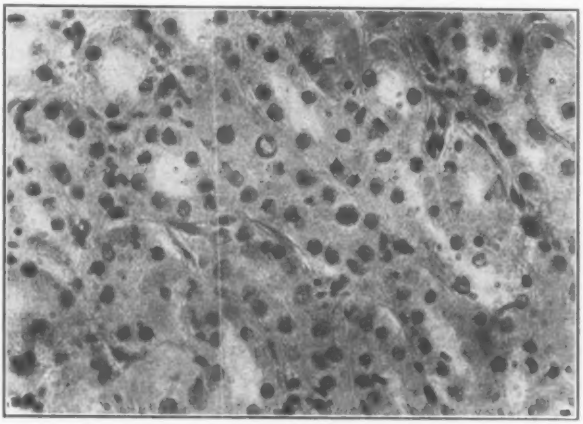

FIG. 3 (×350).—Giant nuclei in regenerated renal epithelium. Experiment 4. Guinea-pig 03.

Characteristic red blood cells were never found in the tubules, but in some specimens shadows of them seemed to be present in some of the capsules of the glomeruli and in few tubules. In the more acute forms of experimental lead-poisoning, hemoglobinuria associated with hemorrhage seems to be the usual results; but in chronic lead-poisoning one is hardly justified in using the term hemorrhagic nephritis in the way that it is employed by Hoffa.[9]

In the majority of the experiments these were the only lesions found even in animals which had been under the influence of lead for two years and over (see, for example, experiments Nos. 22, 23, and 24), and even in those cases in which small areas of cellular infiltration are noted in the record it is very doubtful whether these can be ascribed to the lead-poisoning, because such lesions are not

uncommonly found in the kidneys of untreated apparently normal guinea-pigs.*

This leaves six animals only (Experiments Nos. 11, 12, 16, 18, 25, and 27) that show more definite interstitial changes, and in four of these they are very slight indeed. The kidneys in these four cases did not show any gross lesions, and microscopically a few small areas were encountered in which small groups of tubules (probably adjoining sections through one and the same tubule) were in a state of collapse, and in which the connective-tissue between these tubules was very slightly proliferated. Even in the remaining two animals the condition is by no means striking.

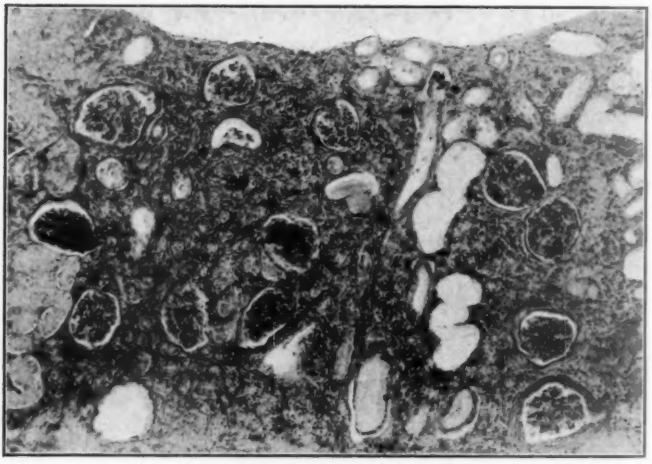

FIG. 4 (× 100).—Area of chronic nephritis with collapse of many tubules, dilatation of others, and capsular thickening of glomeruli. Experiment 27. Guinea-pig 69.

In Experiment 18 it is described as follows: "The kidneys show just visible, fairly regular pitting of the surface," and in Experiment 27, "kidneys fairly large, capsule non-adherent, surface coarsely and finely dimpled."

The most marked lesion found in the latter case[27] is shown in Fig. 4. The photomicrograph gives an idea of the slight depression on the surface, which is largely due to the collapse of the tubules. The slight thickening of the connective-tissue between them is also clearly brought out. At the base of the picture there is a moderate infiltration of the tissues with lymphocytes near some large vessels. The picture also shows a corresponding enlargement of other tubules in the vicinity, which is the usual thing in such cases.

* See Ophüls. Occurrence of Spontaneous Lesions in Kidneys and Livers of Rabbits and Guinea-pigs, Proc. Soc. Exp. Biol. and Med., 1911, viii, 75.

The epithelium in these dilated tubules is partly atrophied, but in some the epithelial cells are evidently enlarged and proliferated.

The glomeruli in this figure are also of some interest, because they show the only lesion which was ever observed in them, and which occurs practically exclusively and constantly in foci of this character, namely, a fibrous thickening of the capsules which is usually rather moderate in extent and may be associated with a slight proliferation of the capsular epithelium.

None of the specimens show the slightest lesions in the blood-vessels.

The condition even in the most advanced case is in no ways similar to what one finds in the kidneys in human cases of chronic lead-poisoning. In them the involvement of the bloodvessels and of the glomeruli is a striking feature of the process, so much so that to all appearances the vascular lesions are primary, the kidneys in many cases being indistinguishable histogically from other forms of arteriosclerotic nephritis.

In a general way the lesions in the guinea-pig resemble histologically those observed in uranium nephritis in the same animals, although they are by no means so severe nor so extensive.

The reports in literature in regard to the action of lead on the kidneys of animals are very contradictory.* On the one hand we have Charcot and Gombault's[7] well-known report, according to which they found "granular" kidneys in 7 out of 15 guinea-pigs after continued administration of carbonate of lead by mouth. They claim that histologically the alterations found were identical with those in human lead nephritis. Their experiments lasted from two days to one year, and in one instance "distinctly granular" kidneys were found as early as the seventeenth day (Experiment 10). None of the later investigators have been able to repeat these experiments so successfully, if we except a rather general statement by Provost et Binet,[12] who after some rather unsuccessful preliminary attempts used Charcot and Gombault's method of feeding carbonate of lead to guinea-pigs on 25 animals; 17 of these survived the first month, most of them dying between the second and third months, One animal only lived six months. They say "lésions renales très accentuées chez la plupart d'entre eux, surtout quand l'intoxication avait duré longtemps," and make a similar statement in regard to fourteen rats that were treated similarly. It is to be regretted that they do not give their observations in greater detail. The experiment of Paviot[18] with carbonate of lead on six rabbits in which he found no gross lesions, but microscopically what he calls extensive sclerosis in two animals after about six months, are no more convincing, to my mind, especially as such lesions very commonly occur spontaneously in rabbits. What is particularly suggestive in this regard is the fact that the lesions in

* At the end of the paper there is a list of the more important articles in historical sequence.

his cases appear to start in the pyramids and extend into the cortex radially as the spontaneous lesions in rabbits are likely to do.

Coën and d'Ajutolo,[11] who experimented on rabbits with acetate of lead for at most 153 days, found only slight interstitial lesions, but believe that a longer administration of the poison might have produced a true renal cirrhosis, a surmise which hardly seems to be justified in the light of our experience. Jores,[26] who also used rabbits, found one small scar only in a rabbit that had been under experimentation for fourteen months and twenty days. All others who have paid any special attention to this point (Hoffa,[9] Oliver,[13] Stieglitz,[14] Annino,[16] Hirsch[20]) describe lesions no more marked than those which were observed in our own experiments.

The conclusions, therefore, seems to be justified that in the warm-blooded animals so far experimented on (guinea-pigs, rabbits, rats, cats, and dogs), chronic lead-poisoning produces in the kidneys primarily epithelial lesions, which condition eventually may be followed by a certain type of granular atrophy, due to collapse of groups of tubules, associated with some fibrous thickening of the connective-tissue between these tubules and of the capsules of the glomeruli in these areas, but that these changes always remain quite limited and are by no means constant. On the basis of my experiments I do not believe, as Jores[26] does, that these later lesions are at all more common in guinea-pigs than they are in rabbits. Certainly nothing has ever been reproduced in any of the animals mentioned which in any way resembles human lead nephritis, Charcot and Gombault's[7] statements to the contrary notwithstanding.*

The existence of epithelial lesions is acknowledged by all investigators, but even in this regard there are to be found certain minor discrepancies in the various reports concerning the exact form of degeneration of the protoplasm and of the nuclei of the cells involved. Annino[16] claims never to have seen any mitoses, which is certainly quite remarkable.

One curious feature in our experiments is the fact that whereas many of the other investigators have described calcareous deposits in the uriniferous tubules, often in large numbers, we have not encountered them in the kidneys of any of our animals, in spite of the fact that they were looked for carefully. They were first observed by Charcot and Gombault,[7] who say in regard to them, "On the cut surface one finds, especially in the pyramid, a large number of whitish, highly refractile dots and lines made up of solid masses of calcareous material, which infiltrates the tubules," and

* The statement which is sometimes found in literature that Leyden reported successful experiments of this character is entirely erroneous. In his paper read before the Verein f. innere Medicin at Berlin, March 19, 1883, and published in the Deutsch. med. Woch., 1883, ix, 185, Leyden reports a case of human lead nephritis, but makes no mention of any experiments whatever, nor does his pupil Muschold in his dissertation, Die Bleivergiftung eine Ursache chronischer Nierenerkrankung, Berlin, 1883.

in another place, "One finds a certain number of the narrow branches of the loops of Henle filled with solid calcareous masses, the cellular structure of which becomes evident after decalcification." They are prominently mentioned and discussed by Prevost and Binet,[12] whereas Stieglitz[14] found them only rarely. Hirsch[20] and Hoddick[27] also mention them, others do not seem to have paid any special attention to this point. Paviot[18] did not find them in his few experiments on rabbits, and believes that Charcot and Gombault's[7] experimental results are largely due to the blocking of the tubules by the calcareous masses. These deposits are of great interest on account of the apparent close relation of chronic lead-poisoning in man to gout. The occurrence of similar calcareous deposits, containing urates also in the pyramids of the kidneys, is very common in such cases in man, and these sometimes are the only manifestation of the existence of a gouty diathesis. Whether the deposits in the animals also contained urates does not seem to have been ascertained. I may state here in passing that in spite of special attention to this point no evidences of gout was ever detected in any of our animals.

The reports in regard to glomeruli and bloodvessels are also most curiously conflicting, which in itself suggests that they are by no means well marked or constant. A hyaline degeneration of the glomeruli has been described by Coën and d'Ajutolo[11] and by Stieglitz,[14] which we cannot confirm in our specimens. It is often difficult to make out exactly what sort of changes the authors allude to, because their statements are very general and few of them give any illustrations of the lesions encountered; but at best the lesions described appear to be slight, somewhat like those described above in our own specimens (slight fibrous thickening of capsules of Bowman, possibly associated with some atrophy of the glomeruli themselves, and a moderate proliferation of the epithelial lining of the capsules), and they are in no way comparable to the glomerular changes observed in human nephritis, either of the arteriosclerotic type or in well-marked glomerulonephritis.

So far as the bloodvessels are concerned, much emphasis is usually placed in reviews of the literature on Annino's[16] work, probably on account of a notice in the Virchow-Hirsch Jahresberichte (1894, I, 375), stating that he found "severe endarteritis which even lead to complete vascular obstruction." If one reads the original, however, and particularly the description of findings in the individual experiments, one discovers to his surprise that in most organs the arterial lesions were very slight indeed, possibly non-existent, except in the lungs, where Annino actually claims to have seen an endarteritis tending to occlusion.*

* Annino[16] used 9 animals altogether in his experiments, 2 young dogs, 3 rats, 2 rabbits, and 2 guinea-pigs. The animals were observed for a few months only, one of the dogs lived longest, for seven months. None of them showed any important gross lesions.

Similar conditions in the pulmonary vessels have been noted by Stieglitz,[14] Greven,[25] and Jores,[26] but Jores very correctly pointed out that normally these vessels in rabbits, and he might have added in guinea-pigs, have very thick walls and a very narrow lumen, and that normally also the intima may be somewhat thicker and more cellular in places. Jores[26] still believes that he found a thickening of the intima which exceeded the physiological limits in those of the animals which had been exposed to the influence of lead longest, but even so he believes these slight alterations to be unimportant, and at the end of his paper he emphatically denies the importance of the vascular changes described by the above mentioned authors, and also by Maier and his coworkers. I fully agree with him on this point and wish to state positively that neither in my specimens have I ever seen anything resembling arteriosclerosis, nor am I at all convinced, from a careful study of the literature, that such changes have ever been produced in animals by chronic lead-poisoning.

The heart, so far as its weight is concerned, and the aorta were found to be entirely normal in all my animals.

THE LIVER. The lesions in the liver in chronic experimental lead-poisoning have attracted comparatively little attention, and yet from the results of my experiments it would appear as if they were really more important than those which occur in the kidneys.

Coën and d'Ajutolo[11] seem to have been the first to pay some attention to the liver. They describe primarily a "necrotic, granular degeneration of the liver cells," and later a chronic hyperplastic periangiocholitis. Since the latter, however, is commonly observed spontaneously in rabbits,* which animals they used for their experiments, this latter condition was possibly not the result of the lead-poisoning.

Oliver,[13] who also used rabbits, makes the following statement: "In the liver of animals and in the liver of lead-workers there will be found an atrophied, cloudy, or granular condition of hepatic cells, amounting at times to a true fatty degeneration. The columns of cells become irregular and slender, and thus become separated from each other by spaces of considerable size. For nearly all the specimens taken from human beings and ruminants these spaces between the individual hepatic cells and rows of cells were occupied by small round cells. There was an increase of connective-tissue elements, derived in all probability from the capillaries or the sheath which surrounded them. In other words, there was an intercellular cirrhosis not unlike that met with in congenital syphilis. The capillaries of the lobules were generally found to be overdistended, a condition observed nearer the centre than the periphery. Interstitial inflammation with degeneration of columns of

* Ophüls, loc. cit.

liver cells was so generally present in all specimens that I cannot but regard this as a constant lesion, and as one likely to arise in the early stages of lead-poisoning." In the accompanying plates he shows the microscopic picture of a liver with well-advanced cirrhosis in a girl, aged eighteen years, who had worked only forty days in a lead-factory. His Fig. XXVIII, showing the microscopic picture of the liver of a rabbit with lead-poisoning, does not show any resemblance to the changes observed in our animals.

Laffitte[15] emphasizes the hepatic changes in chronic lead-poisoning more strongly than any other author. He gave carbonate of lead to four rabbits by mouth for from one and one-half to five months and found no epithelial lesions except atrophy, but marked early interstitial lesions resembling those of cirrhosis. He further presents six human cases of cirrhosis in chronic lead-poisoning, two of which are personal observations. What has been said above in regard to spontaneous lesions in rabbits naturally applies here.

Annino[16] also describes degenerative lesions in the liver and some perivascular cellular infiltration and proliferation of the periportal connective-tissue.

Jores[26] found hematogenous pigmentation to a varying degree and marked hyperemia in the liver of his rabbits, but no other important changes.

The following interesting statement is made in Mallory's recent book on *Principles of Pathological Histology*:* "Lead salts given to rabbits and other animals in amounts just below the minimum fatal dose will produce in a few days a hyaline lesion in the liver cells similar in all respects to that found in the alcoholic cirrhosis of man. Some of the cells undergo necrosis and are removed by the digestive action of leukocytes. Regeneration of liver cells is shown by mitotic figures. This observation suggests that the so-called alcoholic cirrhosis of man may possibly be due to lead."

This is by far the most accurate observation so far recorded in literature.

It is astonishing that the severe gross changes in the liver which follow later in the stages of chronic experimental lead-poisoning and which may be well developed at the end of nine months (see our observation to 14) have apparently never been recorded before.

The initial lesion which has been constantly found in all our cases in which a microscopic examination was made is, as Mallory states, a peculiar hyaline degeneration of the protoplasm of the liver cells associated with pyknosis of the nuclei, eventually terminating in necrosis. The change is clearly shown in three cells in the centre of Fig. 5. The degenerated cells occur either singly or in small groups of two to three cells without any characteristic distribution so far as could be made out. Eventually the remnants of the cells

* F. B. Mallory, The Principles of Pathological Histology, Philadelphia, 1914, p. 507.

are either absorbed or may be removed by leukocytes, as stated by Mallory. New cells to take their place are produced by mitosis.

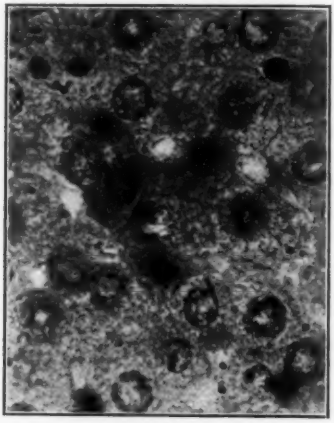

FIG. 5 (× 750).—Hyaline degeneration of small group of liver cells with pyknosis of neuclei. Experiment 3. Guinea-pig 02.

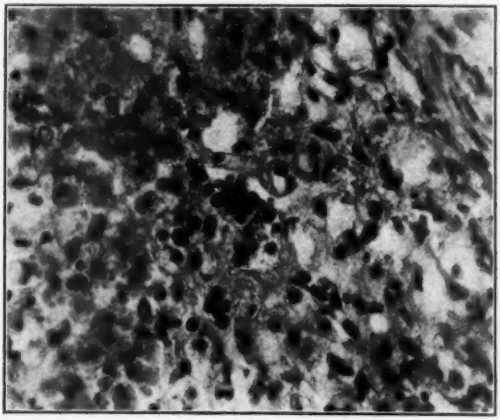

FIG. 6 (× 350).—Fatty degeneration and necrosis of large central group of liver cells with beginning collapse of tissue. Fibrous thickening of capsule. The cells with deeply staining nuclei in centre of field are situated in the central vein of a lobule. Experiment 14. Guinea-pig 3.

In the end these degenerative lesions which may be associated with fatty degeneration may become more and more extensive, so much so as to destroy large parts of the liver tissue, as shown in

Fig. 6, in which practically all the cells in the field are either in a state of severe degeneration or actually necrotic and disintegrating.

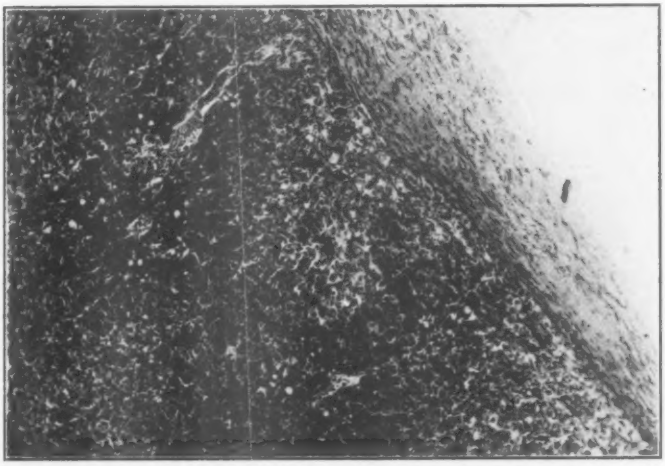

FIG. 7 (× 100).—The same specimen taken with a low power, showing distribution of lesion and marked chronic perihepatitis. Experiment 14. Guinea-pig 3.

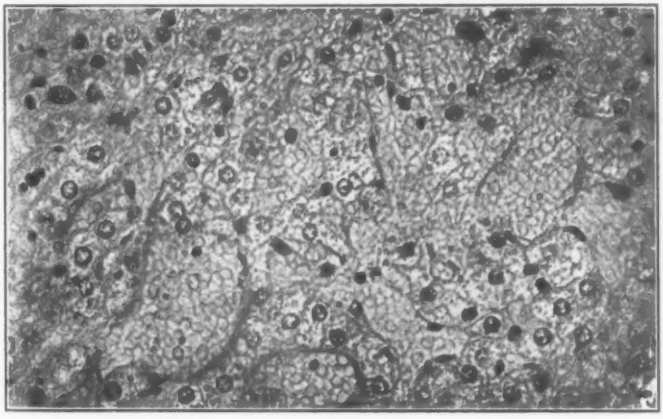

FIG. 8 (× 350).—Dilatation of capillaries due to atrophy and necrosis of liver cells. Experiment 16. Guinea-pig 1.

The removal of the dead liver cells naturally causes a tendency to collapse on the surface, and in the interior of the organ a tendency to dilatation of the capillaries in the same way, as one observed it in the red parts of a human liver in subacute yellow atrophy.

Fig. 7 is a photomicrograph of the same lesion shown in Fig. 6 at a lesser magnification, and brings out more clearly the peculiar localization of it. The dilatation of the capillaries in the interior

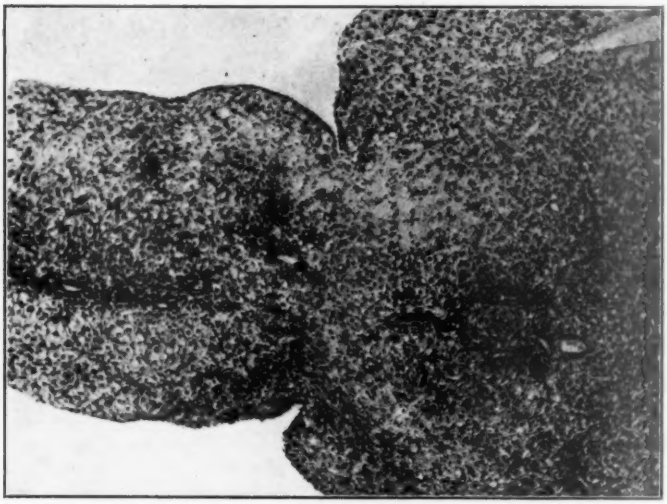

FIG. 9 (× 100).—Extensive areas of degeneration and necrosis of liver cells with dilatation of capillaries and deep retractions due to collapse. Experiment 22. Guinea-pig 33.

FIG. 10.—Gross appearance of liver of guinea-pig 54 (Experiment 21), showing irregular fibrous thickening of capsule, finely granular surface, and large irregular depressions.

of the organ is well shown in Fig. 8, whereas the tendency to collapse is clearly observed in Fig. 9, in which there are also some large areas of capillary dilatation.

The condition is similar to subacute yellow atrophy. Sometimes it involves the organ diffusely, as in observation No. 14, in which the liver remained fairly smooth; but more commonly it is localized in circumscribed spots, and in this way produces first a more or

FIG. 11.—Gross appearance of liver of guinea-pig 35 (Experiment 17), showing finely granular surface and dimpling.

less regular pitting of the surface. Later there is a markedly granular appearance with shrinkage, which in the gross strongly suggests cirrhosis (Figs. 10 and 11).

There is this important difference, however, that there is little if any proliferation of the connective-tissue associated with the process, at least in guinea-pigs. Fig. 12 is taken from the specimen

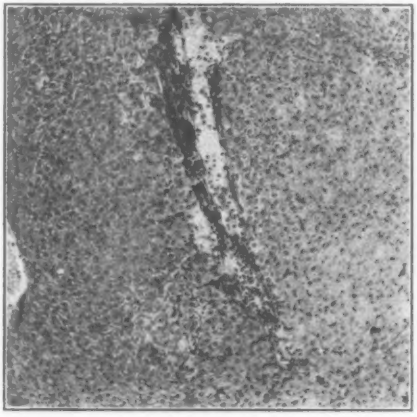

FIG. 12 (× 100).—Cellular infiltration and fibrous thickening of periportal connective tissue. Experiment 20. Guinea-pig 24*a*.

in which the interstitial lesions were most clearly developed in an animal which had survived for one year and eleven months (observation No. 20). The later animals (Nos. 21, 22, 26), in all of which the hepatic lesions were well developed, showed less proliferation

in the periportal connective-tissue, which in observations No. 22 (two years and two months) and No. 26 (three years and one month) amounted to no more than a slight cellular infiltration. It does not appear to be likely, therefore, that the lack of connective-tissue formation was due to insufficient time for its development or that a prolongation of the experiments, if possible, would have brought more striking results in this regard.

Kretz* defines cirrhosis as a "focal recrudescent chronic atrophy modified by intermittent degeneration of the parenchyma." It appears to me that his definition applies much more correctly to the changes observed in these guinea-pigs than to cirrhosis in man, and that it seems evident that such a process cannot in itself explain the abundant connective-tissue formation in human cirrhosis. And yet the process in our experiments would seem to be specially suitable to reproduce conditions resembling human cirrhosis because it is associated with severe anemia, a combination to which Kretz* also alludes in his paper. It is worthy of note that in spite of the constant occurrence of degenerative lesions of moderate degree in the livers of all guinea-pigs examined, and of slight pitting in all animals after the ninth month, severe changes were found to be comparatively rare. They were present in Cases Nos. 14, 16, 17, 20, 21, 22, 26 in seven out of twenty-eight animals. Of the three cases which lasted over three years, one only showed advanced changes in the liver.

The following is a short *resumé* of the more interesting experiments:

14. Guinea-pig 3. The liver is much reduced in size, measuring 5 x $3\frac{1}{2}$ x 2 cm. (normal measurements at least 7 x $4\frac{1}{2}$ x $2\frac{1}{2}$ cm.). The capsule is slightly thickened diffusely, and in places there is a very marked fibrous thickening of the same (Fig. 7). The surface of the liver is smooth, but the shape of the organ is much distorted. The sharp edges are all rounded, and here and there along the anterior now rounded border one sees rather coarse nodules with fairly deep rounded depressions between them. The microscopic appearances of the liver tissue are shown in Figs. 6 and 7. There are many large areas of fatty degeneration and necrosis in which one finds considerable granular hematogenous pigment. The periportal connective-tissue is cellular, slightly increased in amount, and sends slender projections to the thickened capsule. The spleen is large, over twice normal size; marked perisplenitis; few drops of clear fluid in abdomen; chronic pericarditis.

16. Guinea-pig 1. The liver is slightly reduced in size ($5\frac{1}{2}$ x $4\frac{1}{2}$ x $2\frac{3}{4}$ cm.). The capsule is slightly thickened. The surface is finely and evenly granular, showing dark-red spots about 1 mm. in diameter with a grayish depressions between them. On the lower surface

* Lebercirrhose, Verh. der Deutsch. path. Ges., 1904, viii, 54.

of both lobes there are some deep irregular fissures. Microscopic sections show large areas of fatty degeneration and necrosis with marked dilatation of capillaries (Fig. 8) and areas of collapse on the surface of the liver. No hematogenous pigment was found in the liver. The periportal connective-tissue is slightly thickened and infiltrated with lymphocytes. The spleen was of about normal size. Marked, slightly hemorrhagic, ascites; chronic pericarditis; serofibrinous pleurisy.

17. Guinea-pig 35. The liver is of nearly normal size, measuring 7 x 5 x 2½ cm. The surface is distinctly granular (Fig. 11). The granules measure about 1 mm. in diameter. In addition there is some deep pitting, more especially on the lower surface of the liver, where there are many deep fissures. Sections show large areas of fatty degeneration and necrosis, with collapse of tissue on the surface, causing the granular appearance and the pitting. Slight cellular infiltration of the periportal connective-tissue; slight perihepatitis; marked ascites; chronic pericarditis; chronic pleurisy with effusion; anasarca.

20. Guinea-pig 24a. The liver is much shrunken, measuring 5¾ x 4 x 2 cm. The capsule shows a marked irregular fibrous thickening. The shape of the liver is much altered, much like the liver of Guinea-pig 3. The edges are rounded off and the various lobes firmly adherent to one another. The surface shows coarse nodules with deep furrows between them. Sections show large areas of fatty degeneration and necrosis with dilatation of capillaries and collapse of the liver tissue. The periportal connective-tissue is normal except for a slight cellular infiltration. The spleen is large (3½ x 1¾ x ¾ cm.). Marked ascites; hemorrhagic pericarditis; hemorrhagic pleurisy.

21. Guinea-pig 54. The liver is considerably reduced in size, measuring 6 x 4 x 2 cm. The capsule shows a marked irregular fibrous thickening. The surface is uneven and distinctly finely granular, There is also much deep pitting (Fig. 10). Sections show many large central areas of fatty degeneration and necrosis, with collapse of liver tissue and dilatation of capillaries. Much hematogenous pigment is found in the capillary endothelium. In this guinea-pig the cellular infiltration and proliferation of the periportal connective-tissue is more marked than in any of the others as shown in Fig. 12. The spleen is large, 4 x 2½ x 1 cm. Much ascites; hemorrhagic pericarditis; serous pleurisy; anasarca.

22. Guinea-pig 33. The liver is slightly reduced in size, measuring 6½ x 4½ x 2½ cm. The capsule is slightly thickened in a few places. The surface is distinctly granular, the granules measuring about 1 mm. in diameter. There is some deeper pitting on the left side above and below. Sections show fatty degeneration and marked atrophy of the epithelial cells in the centres of the lobules, with dilatation of the capillaries. The granules on the surface are due

to collapse of liver tissue. In the periportal connective-tissue one finds a slight cellular infiltration. The spleen is moderately enlarged, about twice normal size. Little ascites; hemorrhagic pericarditis; serous pleurisy.

26. Guinea-pig 46. The liver is much reduced in size, measuring 6 x 4 x 2 cm. The capsule is much thickened and the upper surface of the liver is attached to the diaphragm. All lobules are adherent to one another and the shape of the organ is much altered, much like the livers of guinea-pigs 3 and 24*a*. The cut surface of the right side shows large areas of necrosis and softening. Microscopic sections show the usual lesions and in addition large bacterial necrosis. The spleen is small, firmly attached to the fundus of the greatly dilated stomach by the thickened capsule. Ascites; hemorrhagic pericarditis; serofibrinous pleurisy.

What makes these changes even more deceptively like human cirrhosis is the constant association in them of severe hepatic disease with accumulations of fluid in the peritoneal cavity and with chronic inflammatory lesions of the serous membranes, more particularly of the peritoneum in the upper part of the peritoneal cavity, which are so commonly observed in human cirrhosis.

The possibility of lead as an etiological factor in human cirrhosis as suggested by Mallory has been discussed by few clinical writers. Aubry reported two cases of Coutenot's in 1865, which in literature are usually recorded twice, both under Aubry and Coutenot's names.[36] In one of these cases a typical cirrhosis was found at autopsy in a girl suffering from chronic lead-poisoning. Laffitte, as stated, has collected six cases of this kind, and Oliver[37] seems to favor the idea that lead is an important factor in certain cases of cirrhosis. Potain[38] also believes that chronic lead-poisoning is definitely though rarely a cause of cirrhosis, and cites some cases in support of his view. He believes that the prognosis of this form is unusually good. It has been suggested that alcoholic beverages are commonly adulterated with lead, still the use of sugar of lead for clearing and sweetening the wines has been long discarded. On the whole there seems to be little clinical evidence so far to connect human cirrhosis with chronic lead-poisoning.

In our service among twenty-five necropsies on painters who had all shown more or less evidence of lead-poisoning, only one case of advanced cirrhosis was observed, and this one in a man who except for some anemia did not show any other evidences of lead-poisoning. In addition there were in this series four cases of slight beginning cirrhosis, which would hardly be suggestive of a more common occurrence of cirrhosis in lead workers. It may be interesting to note here that many of the livers in the more marked cases of chronic lead-poisoning did show some degenerative lesions in the epithelium, resembling those observed in the majority of our guinea-pigs.

As a unique finding in our series of animals, I wish to call atten-

tion to the severe myocarditis observed in Case 28 (guinea-pig No. 12), of which photomicrograph 13 gives a representation. This animal happens to be the one longest surviving (for three years and ten months). There were present in addition in this animal marked degenerative and slight interstitial lesions in the kidneys, moderately advanced lesions in the liver, also some ascites, chronic pleurisy with hydrothorax, and a slight chronic pericarditis with effusion. No similar lesions of the heart muscle were discovered in any of the other animals, even in those with much more marked degrees of pericarditis.

THE SEROUS MEMBRANES. The serous membranes have received even less attention than the liver in chronic experimental lead-poisoning. The frequency of pericardial lesions is commented

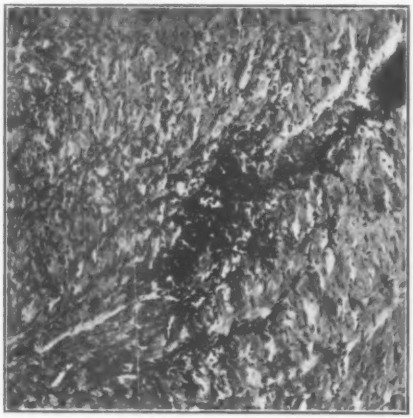

FIG. 13 (× 100).—Myocarditis. Experiment 28. Guinea-pig 12.

upon by Charcot and Gombault[7] in the following statement: "At autopsy of the animals we were impressed by the condition of the pericardium, which we found diseased in 7 out of 15 cases. There was found in the serous cavity an effusion, sometimes simple, sometimes bloody, associated with the formation of abundant false membranes on the surface of the two layers of the pericardium." Their records of the individual cases show that this condition eventually leads to marked fibrous thickening, more especially of the visceral layer, and sometimes to adhesions. They did not observe any changes in pleura or peritoneum except in Observation 9, in which they note the presence of an enormous "blood-clot in the peritoneum," without further discussion. Prevost and Binet[12] also noticed pericarditis in 8 out of 25 guinea-pigs, which were given carbonate of lead by mouth, like the animals in the experiments

of Charcot and Gombault.[7] Stieglitz[14] observed pericarditis in two of his animals. Paviot[18] found a localized gelatinous pericarditis in one of his rabbits. This animal also had been given carbonate of lead in large doses. These are the only references to experimental observations of this kind which I have been able to find in literature and they seem to have attracted practically no attention. Our clinical knowledge in regard to this subject is still more rudimentary. Galvagni* seems to be the only one who has discussed the subject at some length. He says: "In the last year I had the opportunity to observe in my clinic at Modena two cases, which induce me to believe that inflammatory lesions in the peritoneum, more especially in the mesentery, may be a possible result of chronic lead-poisoning." One of these cases came to autopsy, and Foà found perisplenitis; perihepatitis, with total adhesion of the stomach, of the liver, and of the spleen to the diaphragm; total sclerosis of the great omentum; chronic sclerotic mesenteritis with retraction of all loops of the bowel; sclerosis of the solar plexus, etc.

The constancy and extent of the lesions in our guinea-pig is certainly remarkable. Even in the more acute form of the intoxication, especially when the animals receive large doses of lead, small, often hemorrhagic, effusions take place into the serous cavities, as is shown in Experiments Nos. 1 to 5 inclusive. The next series of animals that were given much smaller amounts (Nos. 6 to 11) and also cases 12 and 13, in spite of large doses, show normal serous membranes; but from there on, with the one exception of Experiment 18, the animals all have marked lesions in their serous membranes.

14. Guinea-pig 3 had a few drops of clear fluid in the peritoneal cavity. In the upper part of the abdomen the peritoneum is slightly thickened. The thickening is quite diffuse and somewhat varying in intensity. The capsule of the spleen is thick and fibrous. The parietal pericardium is quite thick and white, and there are some white spots on the visceral layer. The pleuræ are moist, otherwise normal. Smears of the serous membranes show many red-blood corpuscles, many of them nucleated, some megaloblasts, few endothelial cells and lymphocytes, few polymorphonuclear leukocytes. No bacteria were found in spite of the use of various staining methods and in spite of careful search.

15. Guinea-pig 2 had a marked ascites, slight chronic perihepatitis, and slight chronic pleurisy.

16. Guinea-pig 1 had a marked, slightly hemorrhagic, ascites. The peritoneum was slightly thickened, smooth, hyperemic. There were old adhesions between spleen and stomach and between liver and kidney. Slight perihepatitis. Much clear fluid and several thick flakes of fibrin were found in the pleuræ. The lungs were col-

* Galvagni, Sopra un caso di mesenterite in uno saturnino, Riv. Clin., 1884, iii, 23.

lapsed. The pericardium was filled with fluid blood. It was somewhat thickened and the surface slightly rough. Smears of serous membranes showed red blood corpuscles, some lymphocytes, and large phagocytes. No bacteria were found. Hyperemia and edema of subcutaneous fat.

17. Guinea-pig 35 had a slight chronic pericarditis and a marked ascites and hydrothorax, without marked change in peritoneum or pleura, except a slight chronic perihepatitis. Marked general anasarca.

18. No lesions in serous membranes.

19. Guinea-pig 14 had a beginning chronic pericarditis only.

20. Guinea-pig 24*a* had about 10 c.c. of clear fluid with small flakes of fibrin in the peritoneum. There was present a marked irregular thickening of the parietal and visceral peritoneum, including the capsules of liver and spleen. The pericardium contained some slightly hemorrhagic fluid. It was covered with fibrin, and there were some recent adhesions. Much hemorrhagic fluid and much fibrin were found in both pleuræ. Both lungs were collapsed.

21. Guinea-pig 54 had marked ascites. The liver was covered with thin, easily detached shreds of fibrin. Both pleuræ were filled with clear fluid. The lungs were collapsed. Few drops of blood filled the pericardium. Very marked general anasarca.

22. Guinea-pig 33 had few drops of clear fluid in peritoneum. In several parts of the upper peritoneal cavity, especially on the surface of the liver, one noticed thin, non-adherent fibrinous deposits. The peritoneum was also slightly thickened all over. Both pleuræ were filled with clear liquid and the lungs were collapsed. The pericardium contained few drops of pure blood.

23. Guinea-pig 34 had few drops of fluid in peritoneum. The visceral pericardium was slightly thickened.

24. Guinea-pig 31 had little fluid in the serous cavities and a slight pericarditis.

25. Guinea-pig 32 showed but a slight pericarditis.

All three of these animals (Nos. 23, 24, 25) were extremely emaciated females, whereas the strong, well-nourished animals, more especially the males, were the ones which showed the marked lesions in the serous membranes.

26. Guinea-pig 46 had 30 c.c. of slightly bloody fluid in the peritoneum. The peritoneum, especially in the upper part of the abdominal cavity, was moderately thickened. Recent fibrinous deposits were found over the stomach and liver. Thick fibrous bands developed between stomach and mesentery, which had caused a partial strangulation of the small bowel. The duodenum was also surrounded by the adhesions. The stomach above the obstruction was greatly distended. The spleen was attached to the fundus of the stomach and the liver to the diaphragm. The pleuræ were covered with fibrin and contained little fluid. Few drops of bloody

fluid were found in the pericardium. In this case the diseased liver had been invaded by colon bacilli, which had caused extensive bacterial necrosis.

27. Guinea-pig 69 showed nothing but a slight pericarditis.

28. Guinea-pig 12 had a marked ascites, much fluid in both pleuræ, and some fluid in the pericardium. Both the parietal and visceral pericardium were slightly thickened.

The lesions in the serous membranes are evidently closely connected with the lead anemia. As a result of this condition the capillaries become more permeable, not only in the serous membranes, but all through the bodies of the animals, as is shown by the quite common occurrence of a general edema (Experiments 16, 17, 21). In all animals except those which were extremely emaciated the tissues were found not only hyperemic, but distinctly moist. It is possible that the hydremic condition of the blood itself contributes to the liability to leakage from the bloodvessels.

The character of the fluid which exudes into the serous cavities varies greatly, but it is always more or less hemorrhagic and has a tendency to coagulate on the serous surfaces, forming false membranes, which are later organized and lead to thickenings and adhesions. Fig. 7 shows a marked thickening of the capsule of the liver, produced in this way. In Fig. 10 similar capsular thickenings are represented in their gross appearance. In some of the cases the capsular thickening of the liver was so diffuse and well marked that the appearances of a "Zuckergussleber" were closely reproduced.

The danger of adhesions from this source is well shown in Case 26, in which a strangulation of the upper part of the small bowel had resulted from them.

In the pericardium almost pure blood was found quite commonly, and it is interesting to note that this blood was often unusually rich in nucleated reds, and sometimes contained large megaloblasts.

The effusions undoubtedly have somewhat of an inflammatory character, as evidenced by their tendency to coagulation and by the presence in them of rather large numbers of lymphocytes, and occasionally even polymorphonuclear leukocytes. Several specimens were carefully examined for bacteria in smears and culture, but none were found except in the case in which a bacterial infection had been caused as a result of the strangulation.

How far similar conditions occur in human cases of chronic lead-poisoning is still unknown. I have already referred to Galvagni's* case. In our series of 25 painters, pericarditis occurred only twice, both in cases of advanced chronic lead-poisoning. One of the cases showed a typical lead palsy, and both had lead anemia and severe progressive nephritis, marked arteriosclerosis, and some cardiac hypertrophy. In the one case (VIII, 34) the pericardium

* Galvagni, loc. cit.

contained about 200 c.c. of a dark-red fluid with large, red, soft flakes of fibrin. It was covered with a moderately thick layer of grayish-red fibrin, which was firmly attached to the pericardium. No bacteria were found in a culture taken from the pericardium. Peritoneum and pleuræ were normal.

In the other case (V, 50) about one tablespoon of slightly bloody fluid was found in the peritoneum. The pericardium contained about one-half teaspoonful of slightly turbid fluid. Both layers of the pericardium were covered with a thin layer of fibrin which could be detached easily. In the pleuræ over a liter of fluid was found on both sides, the fluid on the left side being slightly hemorrhagic. Unfortunately no bacteriological examination was made of the serous cavities in this last case.

In spite of the slight indications at the present time of a clinical significance of these experimental findings it may very well be that more careful attention directed to the possibility of such occurrences will reveal them much more commonly than would appear at the present time, and the diagnosis of chronic lead-poisoning may clear up some of those puzzling cases of polyserositis or polyorrhomenitis which have been described in literature, especially by Italian clinicians, and may contribute to the proper understanding of such conditions as Pick's pseudocirrhosis and Curschmann's "Zuckergussleber."

CONCLUSIONS. 1. Chronic lead-poisoning in guinea-pigs produces severe degenerative changes in the epithelium of the kidneys and of the liver, leading to necrosis. These destructive lesions are partly compensated for by constant regeneration.

2. In the kidneys these lesions may eventually cause collapse of certain groups of tubules associated with moderate proliferation of the connective tissue and some chronic inflammatory changes. The glomeruli in these foci usually show a fibrous thickening of the capsule. In exceptional instances the defects produced in this way are large enough to cause a somewhat granular gross appearance of the organs.

3. In the liver large areas of collapse occur much more frequently, so much so that marked irregularity of the surface resembling that observed in human cirrhosis is quite common. The connective-tissue proliferation associated with this process is extremely limited. The condition, therefore, cannot well be classified as a cirrhosis, but should be designated as a chronic focal atrophy.

4. Chronic lead-poisoning in guinea-pigs produces general increased permeability of the bloodvessels leading to the production of effusions, usually of a hemorrhagic character, into the serous cavities. These are most commonly observed in the pericardium, but the pleuræ and the peritoneum are often also involved in the process. False membranes are apt to form on the surface of the serous membranes, and their organization leads to the development

of the picture of a chronic polyserositis with fibrous thickening and sometimes adhesions. If this condition is more especially localized in the pericardium there may be the appearances of a pericarditic pseudocirrhosis; if more in the capsule of the liver, that of a "Zuckergussleber" may be reproduced.

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APPENDICITIS AS A SEQUELA OF TONSILLITIS.

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The occurrence of appendicitis as a sequela of tonsillitis has received less attention from clinicians in America and Great Britain than its practical importance warrants. Most of the standard works on medicine and surgery make no reference to it, and it is seldom noticed in current medical literature. This is the more remarkable, since the importance of the tonsil as the port of entry of many forms of systemic infection, and as the starting-point of disease in distant organs, has been well established in recent years in numerous contributions by recognized authorities. In this connection one need only enumerate general sepsis, acute rheumatism, chorea, endocarditis, pneumonia, pleurisy, empyema, meningitis, nephritis, urethritis, orchitis, thyroiditis, and cholecystitis, all of which have been definitely traced to acute tonsillar infections.

About a year ago a patient entered my service in St. Michael's Hospital, a young woman, who developed an abscess in the upper lobe of the right lung a week after an operation for removal of the tonsils during the subsidence of an acute angina.

The relationship between acute rheumatism and appendicitis has

frequently been noted, and when one recalls how often tonsillitis precedes the former, the etiological connection of tonsillitis with appendicitis is more readily apparent. To Sir James Grant, of Ottawa, is due the credit of reporting in 1893, the first modern instance of the association of appendicitis and rheumatism.[1]

The same year Kelynack directed attention to the occurrence of appendicitis secondary to tonsillitis.[2]

In Europe the association of the two diseases has been more systematically studied from both the experimental and clinical stand-points, and consequently more generally recognized by the medical profession as a whole. The similarity in structure between the tonsil and appendix has frequently been pointed out and given as an explanation of their liability to similar infections. While the general trend of opinion among those who have studied the question is to recognize the etiological association of the two diseases, there is considerable diversity of view as to its frequency and the route of transmission of the infection. The experimental and clinical investigation of the latter has elicited much information bearing upon the pathology of appendicitis, especially as to whether the disease is due to a local infection from the bowel or to a hematogenous infection, with secondary localization in the lymphoid tissue of the appendix.

Adrian, in 1901,[3] succeeded in producing appendicitis in rabbits by the intravenous injection of staphylococci, streptococci, pneumococci, B. typhosus, B. coli, B. anthracis, and the tubercle bacillus. He regards the appendix as a point of election for the localization of general infections.

Subsequently, Tedesco[4] investigated experimentally the significance of tonsillar infection, using streptococci, staphylococci, and B. anthracis. He concluded that pyogenic germs from the pharyngeal ring can in rabbits lead to embolic processes in the parenchyma of the appendix, and that it is therefore possible for hematogenous infection to produce characteristic follicular disease of the appendix.

Mori[5] concludes that appendicitis may originate by way of the blood channels.

Kretz[6] believes that almost every case of appendicitis is in causal connection with angina through hematogenous infection.

The conclusions of these authorities have been called in question

[1] New York Med. Record, 1893, I, 609.

[2] A Contribution to the Pathology of the Vermiform Appendix, London, H. K. Lewis, 1893.

[3] Mitteilungen aus der Grenzgebiete d. Med. u. Chir., Band vii, S. 407.

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by Ghon and Namba[7] as the result of their experiments, as well as by Aschoff,[8] Oguro,[9] and others.

Other more recent investigations and clinical reports, however, recognize the possibility of hematogenous infection and the importance of tonsillitis in relation to appendicitis.

Schroetter[10] thinks that an etiological connection between angina and appendicitis has been established, and that this is confirmed by the prevalence of appendicitis a few weeks after the changeable seasons, when angina and throat troubles are especially common.

Kelly[11] recognizes the relationship of appendicitis to tonsillitis, and states that in the surgical clinic of the Johns Hopkins Hospital there were 3 instances of its occurrence out of 91 cases of simple acute appendicitis.

Very convincing evidence in favor of the hematogenous origin of appendicitis has been brought forward by Poynton and Paine,[12] who believe that they have furnished almost conclusive proof that appendicitis may result from streptococcal invasion through the blood stream from a follicular tonsillitis.

Oguro thinks there is no doubt that hematogenous infection does occur in some cases, but less frequently than Kretz maintained. Boit and Heyde[13] express a similar opinion.

Heile[14] does not deny the possibility of hematogenous infection, but it seems to him more natural and simple to assume that the infective agent reaches the appendix and peritoneum from the intestines.

Haeberlin[15] thinks there is no doubt that hematogenous infection occurs after angina and scarlet fever, but that the number of cases in which it plays a role is very limited.

Heyde[16] reports a case of appendicitis following angina, and while he believes a hematogenous origin is uncommon, yet the importance of angina as a predisposing factor should be recognized.

Rèthi[17] says that recently it has been recognized that the tonsils comparatively often form the starting-point of general infections,

[7] Zur Frage über die Genese der Appendicitis, Beitr. z. Path. Anat., 1911, Band lii, S. 120.

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[10] British Med. Jour., 1907, i, 534.

[11] Appendicitis and Diseases of the Vermiform Appendix, 1909, p. 150.

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[14] Zur Pathogenesis des Appendicitis, Arch. f. klin. Chir., 1909, Band xc, 190.

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including appendicitis. An instructive case is mentioned by Deaver of an appendicular abscess following an attack of diphtheria. L. Herdelet[18] reports two cases of appendicitis following tonsillitis. Many other cases might be referred to, but sufficient data have been brought forward to indicate the general trend of opinion, and the wide recognition of the association of tonsillar infections with appendicitis which has occurred in recent years.

Tedesco[19] says he has produced embolism and necrosis of the appendicular follicles experimentally in rabbits. He further states that if appendicitis is immediately preceded by tonsillitis, rapid necrosis of a follicle in the appendix, with perforation or gangrene, is to be anticipated, and therefore operation should be performed early.

Tedesco and also others have referred to the tendency which these cases following tonsillar infection have to develop extremely acute symptoms.

He says that when a staphylococcic infection becomes generalized it is likely to prove fatal. This, however, is by no means an invariable result.

It would therefore appear to be established on experimental as well as clinical data that a hematogenous origin of appendicitis does occur, a fact of much importance in explaining the unusual course at times taken by the latter disease. The clinical relationship of appendicitis and tonsillitis, however, does not depend for its acceptance upon the theory of the hematogenous origin of the appendicular infection. It has been shown that appendicitis can be produced in rabbits by feeding pathogenic bacteria (Chastenet),[20] so that the possibility of infection from the tonsil by way of the alimentary tract is readily apparent.

Some seven years ago my attention was first directed to the occurrence of appendicitis as a sequela of tonsillitis by a case which came under my observation, the history of which is briefly as follows:

H. T., aged nineteen years, student. Father and mother both living and well, aged seventy-five years. They had ten children, all living and well. One brother and one sister had suffered from stomach trouble, with much distention at times, the brother having had a gastro-enterostomy performed by Dr. Stiles, of Edinburgh. The patient, though not robust, had always had good health.

On January 10, 1907, he felt chilly, "out of sorts," had slight headache, general pains, anorexia, and sore throat. When I saw him the next day, examination showed that the tonsils were enlarged and that there was follicular inflammation. The pharynx, fauces,

[18] Gaz. Hebd. Soc. Méd. de Bordeaux, April 12, 1914 (British Med. Jour., June 6, 1914).

[19] Wiener med. Wchnsch., January 9, 1914, p. 82.

[20] Thèse de Paris, 1897.

soft palate, and uvula were also swollen, dusky red in color, and there was a slight sticky exudate on the pharynx. Temperature, 102°; pulse, 100.

Rest in bed, gargles, and aspirin were prescribed. A culture taken from the throat showed Staphylococcus pyogenes aureus.

On the following day the condition of the throat was considerably better; the temperature was 102.5° and the general condition remained much the same. On January 13, I was called to see him at 4 A.M., owing to the development of severe epigastric pain, gastric distention, and belching of gas. There was some tenderness over the epigastric and left hypochondriac regions, and the stomach was so distended that the outline of the great curvature was plainly visible, moving up and down with respiration. The bowels had been moving freely. Temperature, 100°; pulse, 84. There was absolutely no pain, tenderness, distention, or rigidity over other areas of the abdomen. The throat condition had greatly improved. The pain disappeared in a short time, and for two days the patient was apparently much better; the throat had cleared up. Evening temperature, 98.1°; pulse, 80.

On the evening of January 15, there was a recurrence of pain, and he was sent to the hospital on January 16. On admission, at 10 A.M., the temperature was 98°; pulse, 84. Gastric distention, with retching, but inability to vomit, had recurred, and the patient looked ill, out of proportion to his other symptoms.

Dr. G. A. Bingham saw him in consultation at 4 P.M. The temperature was 97.3°; pulse, 84; respirations, 18. There was distention in the epigastric region, but no pain, tenderness, rigidity, or distention elsewhere. He considered operation unwarranted. The throat was practically well.

During the night general abdominal pain, tenderness, and distention developed. The temperature increased to 99.2°; the pulse to 140. The condition unfortunately was not reported to me until my arrival at the hospital in the morning. The patient at that time had all the signs of a generalized peritonitis. An operation was performed and a gangrenous appendix found, with a generalized peritoneal infection. The patient died the same evening. No autopsy was obtained.

Urinalysis on the morning of operation was negative; leukocytosis, 30,000.

The case was so unusual in its course, so distressing in its outcome, and presented so many puzzling features, that I have since followed the subject with much interest.

Dr. Lorne Main, of Dundas, has kindly supplied me with notes of another case, as follows:

J. W., aged twenty-two years. The patient was a robust young man, weighing 190 pounds. He had had an attack of gonorrhea three years previously and a syphilitic infection one year before the

illness to be discussed. On March 4, 1911, he sought treatment for acute follicular tonsillitis. His temperature was 103.5°; pulse, 96. He complained of slight pain and tenderness in the right iliac region, which had, however, disappeared by the next day. Under the usual treatment his temperature became normal on March 8, although he was not allowed out until March 14. He felt very well, and went out for a walk. The tonsillitis had entirely subsided. About 9.30 in the evening he complained of headache and feeling tired; the temperature was normal; pulse, 80. On the next day, March 15, he was about the house; pulse and temperature both normal. The bowels had not moved for two days. At 3 A.M. on March 16, he was attacked by severe abdominal pain.

Dr. S. Cummings operated at 3.30 P.M. There was a gangrenous appendix, with what appeared to be gangrenous areas in the adjacent portions of the small and large bowel. The patient died at 1.30 A.M., March 17.

Other observers have reported cases in which the tonsillitis preceded the appendicitis by only a few hours, and it has been noted that rheumatism may develop coincidently with or in some cases follow the onset of appendicitis.

In both these cases a remarkable feature was the latency of symptoms referable to the appendix. The absence of fever or increase of pulse rate until the sudden development of fulminating symptoms may also be noted. In both cases a gangrenous appendix was found within about twelve hours after the onset of symptoms which might be definitely referred to the appendix. In my case the early pain and tenderness were both referred to the epigastric and left hypochrondriac regions, especially the latter, and the stomach showed extreme distention. Dr. Bingham and I both attributed the epigastric pain and tenderness to the extreme distention of the stomach.

Neither Dr. Bingham nor myself could detect either tenderness or rigidity over the region of the appendix a few hours before the symptoms of general peritonitis developed.

In appendicitis my own experience is that whereas pain may be referred to the epigastrium or other areas, yet tenderness on pressure and at least some rigidity over the region of the appendix, are almost invariably to be demonstrated by careful examination.

Unfortunately a postmortem examination was not permitted, but I[21] was of opinion that there was a generalized infection, with involvement of the lymphoid tissue, in both the stomach and appendix to account for the symptoms.

[21] The above opinion was expressed seven years ago. Since this paper was read in July, 1914, Edward C. Rosenow has published his article on the Bacteriology of Appendicitis and its Production by Intravenous Injection of Streptococci and Colon Bacilli, Journal of Infectious Diseases, March, 1915, xvi, 240, in which he proves, experimentally, that such multiple hematogenous infection of the appendix, duodenum, small intestine, and stomach may occur.

Another possible explanation of the early gastric symptoms is that they were due to a generalization of the infection from the tonsil with secondary involvement of the appendix, or to an infection of the appendix by way of the alimentary tract; in either case the acute early process subsiding with coincident improvement for some days of the pulse, temperature, gastric condition, and general symptoms. After smouldering in the appendix the infection suddenly became active with the onset of fulminating symptoms.

In Dr. Main's case there was definite evidence of involvement of areas in the small and large intestine, as well as of the appendix, suggestive of a hematogenous infection. The evolution of symptoms would suggest that an infection from the tonsils of the lymphoid tissue in other areas of the alimentary tract had occurred, and after smouldering for a time suddenly became active with the onset of fulminating symptoms.

In this case it is interesting to note that the patient complained of slight pain and tenderness in the right iliac region, which, however, had disappeared by the next day (March 5). The acute fulminating symptoms appeared March 16 after the patient was apparently well, suggesting that in the interval the infection in the appendix had remained dormant.

Dr. Arthur Wright has furnished me with notes of the following case from Dr. G. A. Bingham's clinic in the Toronto General Hospital.

Mrs. W., aged forty-seven years, came under observation on April 1, 1914. She had for the previous three days been suffering from pain in the right lower abdomen, radiating to the back. She had previously suffered from acute rheumatism (more than thirty years ago), and two years previously had been treated in the Toronto General Hospital for cardiac trouble. There was profuse uterine discharge, in which gonococci could be demonstrated, and there was also retroversion of the uterus. A parovarian cyst was removed.

On June 10 the patient complained of a sore throat, and on June 12 the tonsils were found to be enlarged and reddened, and there was marked pain on swallowing. On June 16 there was abdominal pain of sudden onset, commencing in the umbilical region, and followed by vomiting. The pain gradually became localized over the region of the appendix, but the temperature and pulse remained normal.

On June 17 the symptoms had become more severe, and pain, tenderness, and rigidity of the right rectus were present to a marked degree, and there was a mass in the appendicular region. Evening temperature 103°; pulse, 114. Leukocytes 21,400. The tonsils were still inflamed.

An operation was performed at 8.30 P.M. and a gangrenous appendix, in a retrocecal position, was removed. Recovery occurred, and on June 26 the patient no longer complained of pain or soreness of the throat.

In conclusion, I[22] would like to emphasize the following points:

1. The importance of bearing in mind the liability of appendicitis to follow acute tonsillitis.

2. That the appendicular involvement may be only part of a generalized infection, hence the gravity of such cases out of proportion to the local symptoms.

3. The tendency for such cases to be atypical in their clinical course, and after smouldering, to suddenly develop fulminating symptoms.

4. Chronic tonsillar infections should be kept in view as the possible cause of similar infections of the appendix.

5. That whereas at least some degree of local tenderness and rigidity are almost always to be elicited on careful examination of the abdomen in the right iliac region in acute appendicitis, in rare cases these signs may be absent.

LIGATION OF THE PORTAL VEIN IN SUPPURATIVE PORTAL PHLEBITIS.

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The problem of attacking pylephlebitis along the lines employed in septicemia of otitic and of uterine origin, has as yet attracted no great attention.[1] A. G. Gerster,[2] commenting on this disease, says: "The evacuation of septic thrombi from the jugular vein in mastoid disease has yielded such excellent results that the application of the principle to the portal vein would be natural and logical. But the anatomical relations, while very favorable in the former instance, are just the reverse in the latter. Only a short piece of the portal vein, that situated in the hepatoduodenal ligament, is approachable. The two mesenteric veins and the splenic are practically inaccessible. Hence, though phlebotomy of the portal trunk in the hepatoduodenal ligament is not impossible, the evacuation of thrombi by flushing through a catheter seems to be too problematic, not to mention the technical difficulties the surgeon might encounter in the closure of the phlebotomy wound."

[22] Read before the Canadian Medical Association, St. Johns, July, 1914.

[1] H. Kehr, Chirurgie d. Gallenwege, 1913. Suppurative thrombi in the portal vein have never been removed operatively. Such a procedure might possibly have a beneficial effect.

[2] Trans. Am. Surg. Assoc., 1903.

More recently, H. Neuhof,[3] in his work on the experimental ligation of the portal vein, has called attention to the following important facts: "Although the portal vein has been more or less completely ligated for accidental injury in a very few instances (possibly two cases), a deliberately planned ligation has never been practised." Further, "The fact that no surgery of the portal vein has as yet developed, appears to depend upon the results of animal experimental studies. Many observers have demonstrated that ligation of the portal vein regularly leads to death in a very short time—in half an hour to two hours."

On the other hand, by experimental work Neuhof has established that gradual occlusion of the portal vein is not fatal. The cases of Brewer and Burdenko[4] indicate that in human beings a gradual occlusion due to pressure, followed by a ligation, is compatible with life, provided collateral circulation has been established. In view of these facts, Neuhof advocated ligation of the portal vein in suppurative pylephlebitis, realizing that the portal thrombus by gradually occluding the portal vein might have induced the development of a collateral circulation prior to the ligation of the vein, and that such a ligation would prevent further bacteria being swept from the radicles of the superior mesenteric vein into the liver. In animal experiments, moreover (Burdenko), it has been shown that the portal vein may be ligated successfully, provided there are preformed anastomoses produced by operative adhesions between the parietes and the omentum and intestines.

Guided by such considerations, and having realized some years ago that an interference at a lower level (*e. g.*, ileocolic vein) would have no effect, the following case was operated upon. The plan of procedure, as soon as the diagnosis was made, was as follows: To insure an adequate collateral circulation, I wished to make, if possible, a venous lateral anastomosis between the left spermatic vein a few inches below its confluence with the left renal vein and one of the larger branches of the inferior mesenteric vein.[5] In this way, the portal and systemic circulations would be satisfactorily anastomosed. In addition, I proposed to perform an omentopexy to establish other anastomotic paths. At the second operation, I planned to ligate the portal vein, to cut the vein above the ligature and drain the hepatic end with rubber tube run into the lumen of the stump and at the same time perform a cholecystostomy to drain the biliary system.

All that I planned to do I was not able to carry out, as will be seen from a perusal of the case report. Owing to the interest of such a case the data will be given in some detail:

[3] Surg., Gyn. and Obst., 1913.
[4] Deutsch. Ztschr. f. Chir., 1913, cxxiv.
[5] The application of this idea to the treatment of cirrhosis might prove of value.

CASE REPORT.—H. G., aged twenty-five years; shipping clerk; married. Admitted February 20, 1915. Died March 1, 1915.

Diagnosis. Acute gangrenous appendicitis. Operation: Appendisectomy.

Postoperative Complications. Pylephlebitis. Secondary Operations: Omentopexy, ligation of portal vein. Cholecystostomy.

Family History. Father and mother dead; five brothers all well; two sisters also well. No tuberculosis or cancer in family.

Past History. Patient smokes ten cigarettes a day. Drinks occasionally. Had gonorrhea three years ago. Denies syphilis, pneumonia, scarlet fever, typhoid, etc. Two weeks ago had grippe and sore throat.

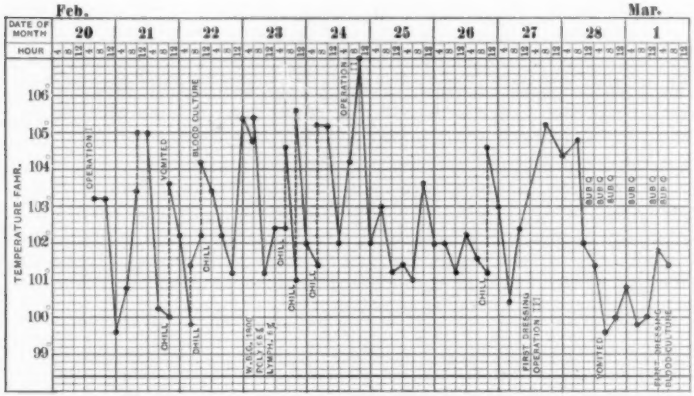

FIG. 1.—Temperature chart.

Present History. Wednesday, February 17, 1915; in the morning the patient experienced general abdominal cramps, associated with vomiting, constipation, chills, and fever. The general pains subsided and became localized in the right iliac region. Patient has been thirsty and has had frequency of urination. Since the day of onset, also very much prostrated, not having had any sleep on account of the pains. Has no cardiorespiratory or renal complaints.

Synopsis. Pain in right iliac region since Wednesday.

PHYSICAL EXAMINATION. *General Condition.* Good. Patient is well nourished.

Head. Eyes react to light and accommodation. No palsies; no petechiæ. Tongue coated. Teeth in good condition. Pharynx congested. Tonsils negative. Ears and mastoid and thyroid negative.

Glands. Negative. *Chest.* Expansion fair and equal. Lungs, anterior and posterior negative; no dulness, no rales. Heart, U. B., third rib; R. B., right border of sternum; L. B., fifth space mid-clavicular line; sounds booming at apex, slight roughness of the first sound; sounds negative at base.

Pulses. Equal, regular, and of good force; tension not increased but rapid.

Abdomen. Symmetrical; tympanitic; tenderness, rigidity, and spasm of right rectus muscle in the iliac region; rebound sign positive. Spleen, kidney, and liver not palpable.

Rectum. Tenderness and sense of resistance in the right iliac fossa.

Extremities. Negative.

Synopsis. Tenderness, rigidity, and spasm of right rectus muscle in right iliac region.

Operative Procedures. *First Operation.* February 20, 1915. (Dr. Shlimbaum, house surgeon) G. and E. Appendicectomy for acute gangrenous appendicitis. Incision two and a half inches, right rectus muscle splitting.

Findings. Appendix found to be entirely gangrenous with fibrinous exudate on tip. No evidence of thrombosis in mesenteriolum.

Procedure. Amputated in usual fashion: stump carbolized, closure of abdominal wound in layers.

Specimen. Last inch of appendix somewhat bulbous, gangrenous and contains foul-smelling pus.

February 21. Looks very septic; two chills. Temperature, 99.8° to 105°; pulse, 106 to 132. Blood culture taken today showed streptococci. Has severe right-sided abdominal pains. Jaundice present.

February 23. Leukocytes, 18,000; polyneuclears, 94 per cent.; lymphocytes, 6 per cent.

February 23. Since operation the patient had chills daily and septic temperature. Conjunctivæ deeply icteric. Complains of pain in right hypochondrium, where there is distinct tenderness over liver, which is distinctly enlarged. Wound clean.

February 24. Jaundice more marked today. Chills and high temperature persist. Tenderness over liver more marked. *Diagnosis:* Suppurative portal phlebitis.

Second Operation, February 24. (Dr. Beer, G. and E.) Patient taken to operating-room with idea of anastomosing inferior mesenteric vein and spermatic (left), and doing an omentopexy so as to establish collateral circulation preliminary to ligation of portal vein for pyelophlebitis.

Incision along the crest of the ilium to left of left rectus muscle. Lower pole of kidney and ureter exposed. Spermatic vein isolated. Vein branch of inferior mesenteric isolated. Attempt at making anastomosis was unsuccessful, owing to very small caliber and thin

wall of inferior mesenteric branch. Peritoneum was then opened to make an omentopexy and portal fissure palpated. Fair-sized indurated area and enlarged glands palpable at portal fissure. Omentum brought down and sutured to parietal peritoneum with plain catgut suture. Peritoneum then repaired as usual. Wound closed in layers.

NOTE. No ascites. Vessels in omentum and branches of inferior mesenteric vein showed little abnormal congestion.

FIG. 2.—Sketch of ligated portal vein and branches with suppurating adherent thrombus proximal to ligature.

February 24 to 27. Patient seemed to remain about same. Temperature was lower and only one chill.

Third Operation. February 27. Ligation of the portal vein for suppurative phlebitis; cholecystostomy for drainage of the bile tract (Dr. Beer).

Incision four to five inches right rectus from ribs to level of umbilicus. Small amount of bile-stained fluid in the peritoneal cavity. Marked venous congestion of the stomach and gastro-

hepatic ligament. As the foramen of Winslow was shut off, and as the distended gall-bladder prevented an easy approach to the region of the portal vein from the right side, the peritoneum over the vein was opened just above the duodenum over what appeared to be the site of the portal vein. Numerous glands of deep red color blocked the approach, and dissection along the tract of the hepatic artery caused very disagreeable bleeding. The bleeding was controlled and a cholecystostomy performed. Then another attempt was made to reach the vein after exposing the choledochus for two inches. This approach was not blocked by glands and exudate, and the large blue vein was found mesial to the duct on a slightly deeper plane. It was impossible to state that there was a thrombus within, though the walls seemed thicker than normal. A ligature was thrown about the vein and, on pulling it sufficiently tight to occlude the lumen, no change in the patient's pulse was detected (Dr. Wilensky). Thereupon the vein was tied with heavy catgut. Rubber dam to ligature coming out along the gall-bladder tube. Wound closed and dressed.

Patient was much shocked and in poor condition when returned to ward at 5 P.M. Under heroic stimulation his condition improved.

February 27, 8.15 P.M. Condition fair. Pulse of fair tension, regular, and of rate of 140. Respiration regular and not rapid (34).

8.45 P.M. Condition unchanged. Tongue very dry, rough and brownish; fur on dorsum.

9.15 P.M. Salt solution not retained by bowel. Answers questions correctly.

9.45 P.M. Condition unchanged. Looks comfortable. Pulse and respiration unchanged. Mind perfectly clear.

10.45 P.M. Pulse 140. Good quality. Beats practically same in force, but occasionally one of weaker quality is felt. Was sleeping. Now awake. Recognizes surroundings and persons previously known to him. Jaundice apparently (artificial light) somewhat less marked. Tongue very dry in centre. Is taking fluids in two to three dram quantities without vomiting. Bile drainage, four ounces.

11.20 P.M. General condition pretty fair. Pulse 134, good quality, regular in rate and rhythm. Does not complain of any pain. Jaundice seems decidedly diminished, particularly of conjunctivæ. Breathing is regular, slightly increased in rate.

February 28 and March 1. Enemata effectual and flatus expelled. Edema of scrotum and lower abdominal wall (perhaps more on left side) present this morning; also of penis and suprapubic region. (Infection?) Retention of forty ounces of urine relieved by catheter.

Lumbar wound infected, fascial necrosis present. Sutures removed, wound opened widely and packed with iodoform gauze. Bile drainage six ounces.

Blood culture taken in morning showed Bacillus coli. In the afternoon patient became gradually comatose, and died forty-eight hours after operation without having given any evidence of blood either in vomitus or in stool.

Urine Report. February 21. Amber; acid; sp. gr., 1028; albumin, heavy trace; sugar and bile negative. Microscopically: hyaline casts, epithelial cells, and a few leukocytes.

February 22. Bile, negative.

February 24. Bile in urine.

February 25. Bile in urine.

February 28. Bile in urine.

Blood Report. February 25. Blood culture of 12 c.c., streptococcus.

March 3. Blood culture; Bacillus coli.

DIAGNOSIS. Acute suppurative pylephlebitis (secondary to acute gangrenous appendicitis). Ligature of portal vein. Streptococcemia.

INCOMPLETE AUTOPSY (by Dr. Baehr). *General.* Marked icterus, no petechiæ. Rigor mortis marked.

Abdomen. The abdominal viscera inspected through surgical incision along the right rectus muscle. No ascites. The portal vein found tied off with catgut about 1.5 cm. above the head of the pancreas. Distal from the ligature, the entire portal system was found to be filled with thick dark brown grumous purulent material consisting of pus mixed with some blood. In the portal vein near the bifurcation and in its left main branch, there were also flat purulent thrombi on the vessel wall. Proximal to the ligature, the splenic and inferior mesenteric vessels were found to be patent. The superior mesenteric and its right colic branch were found to be filled with yellowish white purulent material. In places there were also thrombi. The thrombosed vessels containing yellowish white pus led down to the region of the cecum and ileo-cecal junction. The vessels in the immediate neighborhood of the appendix site were empty and showed no suppurative process.

Gastro-intestinal Tract. Appears normal, showing no circulatory disturbance. The coronary veins along the upper half of the lesser curvature are widely dilated. The appendix stump appeared to be normal.

Retroperitoneal veins behind the ascending colon show a similar dilatation.

Liver. Slightly larger than normal. Its lower border reaches down about one finger breadth below the free border of the ribs. On section the organ is seen to be studded with areas in which there are numerous small yellowish foci consisting of collections of pus. In the immediate neighborhood of each of these areas is also a radicle of the portal vein, filled with dark grayish brown purulent material. The intervening liver parenchyma shows merely cloudy

swelling. The common bile duct is patent and contains some thin yellowish bile.

Gall-bladder. Fastened to the abdominal wall by a catgut suture. A hole in its fundus is also closed with a suture. The organ itself contains a small amount of mucopurulent material which is slightly bile stained.

Spleen. About one-half larger than normally. No organs removed.

Bacterial Report. Bacillus coli from pus from liver.

Microscopic Examination. *Liver.* Capsule of normal thickness. There is moderate passive congestion in the centres of the lobules. The liver cells in this region are somewhat smaller than normal and contain granules of brownish pigment. In Glisson's capsule throughout the organ there are numerous and extensive round cell infiltrations. These have no relation to the bile ducts and the hepatic arteries. In some places, there are large collections of pus cells and cellular detritus, in the midst of which masses of bacteria are frequently to be seen. Most of these abscesses appear to extend from foci in Glisson's capsule. Many of the smaller radicles of the portal vein contain parietal blood-platelet thrombi. Their lumen usually contains red blood cells and only rarely collections of pus cells. The larger radicles of the portal vein appear to be for the most part completely destroyed by a suppurative process and in some places the abscesses can be seen to be extending from this by contiguity.

Spleen. Capsule and trabeculæ of normal thickness. Sinuses in the pulp and the large venus sinuses are markedly dilated. Malpighian bodies rather small, contain no germinal centres and are surrounded by a zone of congestion.

The most striking feature of this case of fulminating portal vein suppuration is to be found in the absence of all signs and symptoms of obstruction to the return flow of the portal blood despite ligation of the portal vein. How this is to be explained I admit I do not know. Whether it was accidental, and due to some anomaly in our patient's circulation, or whether the short duration of the incomplete portal obstruction had forced collateral circulation to such an extent, it is impossible to say at this time. That complete ligation of the portal vein did not lead to some engorgement of the viscera surprised me very much, as I do not believe that the omentopexy gave much assistance to the return flow. Unfortunately the patient's condition became so bad during the third operation that I was not able to do what I had planned, *i. e.*, cut across the portal vein above the ligature and drain the hepatic end with a tied-in tube. Knowing the remarkable power that the liver has shown in fighting infection, I believe that this assistance would be of great value. Further experience along these lines will demonstrate whether one can expect to save these cases in the manner outlined in this paper.

A REPORT OF THREE CASES RESEMBLING PSEUDOSCLEROSIS AND PROGRESSIVE LENTICULAR DEGENERATION.

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The disease known as pseudosclerosis was first recorded in 1883 by C. Westphal. This observer described two cases in which tremor was the most conspicuous symptom. In these cases the symptoms resembled those of disseminated sclerosis in some other respects, but when the brains of these two patients were examined after death, they did not present the usual appearance of the brain in disseminated sclerosis; in one of the cases, however, the brain did show a generalized increase in consistence, but in the other case nothing abnormal was discovered.

Although Westphal seemed fairly certain that the cases he observed were representative of a disease that had not up to that time been recognized, he found it difficult to define its boundaries sharply. The term pseudosclerosis was, therefore, apparently coined to designate the condition.

These observations remained for a long time unnoticed until Strümpell[1] carefully analyzed and described the symptomatology of his own and of Westphal's cases, with the result that he was able to distinguish clearly between, and to separate this condition from, multiple sclerosis.

Similar cases have been recorded by the late Professor Frankl-Hochwart,[2] of Vienna, and also by Fickler[3] and others, but little of importance had been published that could throw any light upon this subject until the observations of Völsch[4] appeared.

Völsch's patient was a girl, aged seventeen years, who had suffered for about two years from pronounced rhythmic tremors of the limbs and trunk and spastic contractures of the extremities, together with dysarthria and dysphagia, and who showed pigmentation of the cornea and of the skin. In this case the postmortem examination revealed marked atrophic cirrhosis of the liver, a condition that had not been diagnosed during life. No abnormality could be detected in the brain. This case was regarded by Völsch[5] as probably one of pseudosclerosis, of the type recognized by Westphal and Strümpell.

It was just at this time (September, 1911) that Wilson's very

[1] Deutsch. Ztschr. f. Nervenh., 1896, xii.
[2] Arb. aus dem Universität, 1903, Heft x, p. 1.
[3] Deutsch. med. Wchnschr., 1904, No. 51.
[4] Deutsch. Ztschr. f. Nervenh., 1911, xlii, 133.
[5] Ibid. 335.

complete descriptions of the disease he termed "progressive lenticular degeneration" appeared.[6] In the appendix to that article Wilson refers to Völsch's case as probably one of lenticular degeneration, and, indeed, this opinion seems entirely justified, in spite of the fact that degeneration of the lenticular nucleus had not been discovered.

Since then similar cases have been carefully studied and recorded: one by Hösslin and Alzheimer,[7] and one by Rumpell.[8]

In the cases that I am about to record here the condition resembled very closely the accepted disease-picture of pseudosclerosis.

HISTORY. The family consisted of two males and three females. Three of the members were affected and two were healthy. John and Annie were admitted to the wards of the Orthopædic Hospital and Infirmary for Nervous Diseases in Philadelphia, under the care of Dr. John K. Mitchell, during the winter of 1913, and at different times subsequently they have been under observation in the outpatient department. Thomas was never admitted to the wards of hospital, but has been under observation in the dispensary.

The patients stated that they came of healthy Irish stock, their parents having been people of good habits; that they themselves had not previously suffered from any disease, and that prior to the onset of their present malady, they had always been able to follow their occupations, John and Thomas as laborers and Annie as housemaid. Venereal disease was denied. John and Thomas admitted that they occasionally used alcohol to excess, but they did not appear to be alcoholic. Annie had always been a total abstainer. Thomas was married, his wife and two children being healthy.

CASE I.—John McC., aged forty-two years. The patient stated that when he was thirty-two years old he noticed "a shaking" of his right arm. He did not feel sick at that time, and declares that he had not been drinking. The shaking became gradually more pronounced, and he soon found that his legs were beginning to feel stiff and that his left hand also was "shaking." About six to ten months after the onset he was compelled to give up work on account of so much "shaking" and stiffness of his limbs; since then he thinks that the stiffness of his legs has gradually grown more and more marked.

Physical Examination.—The patient's expression is abnormal It differs from that seen in paralysis agitans in that it lacks the "fixed" look, and it varies also from the characteristic expression of Wilson's disease in that the spastic and contracted appearance of the facial muscles is lacking; there is, however, a decided tendency to widening of the palpebral fissures, lending to the face an expres-

[6] Brain, 1911.
[7] Ztschr. f. d. ges. Neurol. u. Psychiat., 1912, Bd. viii.
[8] Deutsch. Ztschr. f. Nervenh., 1914, xlix, 54, 161.

sion that might be described as one of astonishment. This peculiarity seemed never to disappear entirely. There was no tremor of the lower jaw. The pupils reacted normally to light and in accommotion and in convergence; the ocular movements were well performed in all directions, and the ophthalmoscopical examination revealed nothing abnormal. Hearing also was normal.

There was a slight regular, continued tremulous movement of the head on the trunk, but there was no evidence of contracture of the muscles of the neck.

While in the standing position the patient presented a marked tremor of both upper extremities, the trunk, and the lower limbs. With the aid of a chair he could stand erect, but quickly became tired. The tremor of the upper extremities was somewhat more pronounced on the right side, and on both sides it was greater in the distal portions. It was a constant to-and-fro rhythmic flexor and extensor movement, which never ceased entirely, and it was always increased by emotion and by muscular effort. Moreover, any strong muscular effort of one extremity seemed to increase the tremor of the other limbs. In performing the finer movements with his hand there was a certain degree of awkwardness that appeared to be due more to increased muscle tone than to the tremor itself; still, spasticity was not very pronounced. The triceps and biceps tendon reflexes were equal and only slightly more marked than normally. Muscular power for all movements of the extremities was about the same on both sides, and was quite normal.

The abdominal and cremasteric reflexes were equal and normal.

His lower extremities were spastic and tremulous. Unlike the upper extremities, the tremor in the lower limbs was more marked in the thighs or proximal portions than in the distal parts. This was pronounced when the patient attempted to walk. Spasticity was fairly well marked, although he did not actually "stab" his toes on the floor when walking. There was no tendency to retropulsion or festination. The patient was able to walk short distances with the aid of a cane, but the effort caused a general increase of the tremulous movements throughout the body and extremities, and, as a consequence, he rapidly became exhausted. At times, because of spasm of the muscles, the right foot was strongly abducted and temporarily everted. The patellar tendon reflexes were distinctly increased, but equally so; the Achilles tendon reflexes also were exaggerated, but equal. Irritation of the soles of the feet caused a prompt flexor response of the toes.

Examination of the thorax and abdomen revealed nothing abnormal. There was no evidence of arteriosclerosis. The systolic blood-pressure was 140. The liver dulness was not diminished.

The Wassermann reaction with the blood serum was negative. The blood count was normal and the urinary examination was negative.

His mentality was, on the whole, good. He did, however, present a certain irritability and lack of initiative, and was somewhat dull; but whether or not his differed from the condition one frequently sees in patients suffering from severe chronic disease is questionable.

There was no discoloration of the skin on any part of the body, and pigmentation of the cornea was also absent.

CASE II.—Annie McC., aged thirty-eight years. The patient stated that she had been healthy until she was thirty-four years old, at which time, she declared, she gradually became more and more "nervous" (she explained that she used the term "nervous," meaning tremulous), and that her limbs became so stiff that she had great difficulty in getting about. She also stated that in less than a year's time after the onset of the tremor she had to give up work. Although at the present time she is not entirely incapacitated, it is only with the greatest difficulty that she is able to go up and down stairs.

Physical Examination. The patient is of average height, but appears to be thin, weighing about one hundred pounds. Her generalized tremulousness is very striking. Her pupils are equal and react normally to light and in accommodation and in convergence. The ocular movements are normal in all directions, and the ophthalmoscopical examination revealed nothing abnormal. There is no pigmentation of the cornea. The cranial nerves all seem to functionate normally; but the facial muscles, particularly on the right side, show a distinct tendency toward spasticity. The widening of the palpebral fissures and the expression of astonishment seen in the other patient are lacking here; nevertheless, this patient's expression, while it does not seem to be entirely normal, is not, on the other hand, characteristic. There is a decided regular tremor of the lower jaw, which is constant, so that the act of puckering the lips is done with difficulty. Her speech is indistinct—not dysarthric, but merely the speech of tremor. Swallowing is performed without any difficulty. There is a slight yet distinct tremor of the head on the trunk, but there are no spasmodic contractures of the neck muscles.

The upper extremities show quite marked to-and-fro flexor and extensor movements, which are more pronounced in the distal portions and equal on the two sides. When the patient stands with the arms hanging at her side a distinct and regular tapping sound is made by her hands striking against her skirt. The arms are slightly spastic. The biceps and triceps reflexes are active and equal on the two sides, but are not increased.

The movements of the extremities in this case, although not greater than those shown in Case I, did present an element of incoördination that was lacking in the previous case, or, at least, was much less conspicuous. At times, when using her hands for the finer movements, the fifth finger and the fourth finger were held in

partial flexion and partial abduction. This resembled, but was not identical with, an athetoid condition.

The muscles of her back are held somewhat rigidly, and there is a tendency for the trunk to be drawn to the right side, as if the deep muscles of the back were in the early stages of a permanent contracture.

The abdominal reflexes are equal, but are difficult to elicit.

The lower extremities are very tremulous and moderately spastic. The patient can walk about the house and go up and down stairs only with great difficulty, and with the assistance of bannisters and cane; she is frequently compelled to stop to "quiet down."

The tendon reflexes of the lower limbs are moderately increased, but equal. There is no Babinski sign, and ankle-clonus is absent. Sensation everywhere is normal. There is no discoloration of the skin. The abdominal and thoracic organs show no abnormalities. The liver dulness is not diminished. There is no evidence of arteriosclerosis. The systolic blood-pressure is 136 mm.

The Wassermann reaction in the blood serum is negative. The blood count is normal and the urinary examination is negative. Mentality is normal.

Case III.—Thomas McC., aged thirty-four years. The patient states that when he was thirty-two years old he first began to feel "shaky." This was at first noticeable only in his hands, but later, he states his legs also began to tremble. He thinks that the tremor is not increasing, but that his legs are getting stiffer.

Physical Examination. The patient appears well nourished and muscular. His pupils are equal and react normally, and his vision is also normal. The eye-grounds were not examined. All his cranial nerves act normally.

His articulation is clear and distinct, and he has no difficulty in swallowing. His expression is normal, and there is no tremor of the jaw or of the facial muscles.

The muscular power of the upper extremities is equal and normal, as are also the triceps and biceps tendon reflexes. There is a distinct regular tremor of both hands. The patient shows a tendency to hold both arms partially flexed at the elbow, and the tremor is not unlike that of paralysis agitans, but lacks the "pill-rolling" position of the fingers so common in the latter disease.

While the tremor does not disappear, it is not increased on voluntary movement. With the patient in the sitting position, scarcely any tremor of the low extremities and trunk is noticed; but when he stands erect a slight regular extensor and flexor movement is noticeable at the knees. While this is not marked, it is, nevertheless, perfectly distinct. The lower limbs are slightly spastic, but locomotion is not markedly impaired. The patellar tendon reflexes and the Achilles tendon reflexes are equal and slightly increased. There is no Babinski sign.

Although the patient's mentality may have been entirely normal, he found fault with physicians, was very irritable, sulky, and sullen, so that reëxaminations and laboratory examinations could not be completed.

SUMMARY. These three patients were all very striking in appearance. They presented tremor of the upper extremities, which began gradually and increased in severity, spreading slowly throughout the body, and involving the lower extremities as well; at the same time a gradually increasing tonicity of the muscles developed, until finally all voluntary movements and locomotion were greatly affected.

Dysarthria was not well defined, but in one case articulation was indistinct. Characteristic mental disturbances were lacking in all.

The diagnosis of these cases, based on purely clinical observation, would seem to preclude the possibility of an existing multiple sclerosis, for the usual symptoms of that condition were lacking—there was no nystagmus, the eye-grounds were normal, and there were no disturbances of sensation. A certain amount of muscular weakness was evident, but it was not greater on one side than on the other, and it did not even approach true paralysis; yet increased muscular tonicity was very general.

The tremor was more violent and of greater amplitude in Cases I and II. It was of a to-and-fro or backward and forward movement, showing some increase on voluntary movement, but being entirely free from the uncertainty of direction that in so many cases of advanced multiple sclerosis exhibit on voluntary movement. The tremor in Case III was much milder, and more closely simulated the regular rhythmic movement of paralysis agitans.

The difference in symptoms presented by these cases to those of progressive lenticular degeneration was indeed indefinite, for the condition appears to have been a family affection, very slowly progressive in character, but not, however, developing, as did Wilson's, in youth. The tremor seems to have been almost identical in character, but the degree of spasticity was slighter, and contractures were not observed, nevertheless, a tendency to tonic muscular spasm was present in one (Case I).

Strümpell[9] has referred to certain similarities that pseudosclerosis bears to Wilson's disease, and points out that in characteristic cases of pseudosclerosis the tremor is more conspicuous than the spasticity; whereas in lenticular degeneration the spasticity and contracture are more marked than the tremor, for it is known that lesions of the lenticular nucleus are capable of producing high degrees of spasticity. Nevertheless, if spasticity and contracture of muscles were sufficiently great, tremor of that part must of necessity grow

[9] Deutsch. Ztschr. f. Nervenh., 1913–1914, l, 455.

less pronounced, for the limb may become partially fixed. This did occur in one of my cases.[10]

Without a postmortem examination it would seem uncertain whether a positive differential diagnosis would be entirely justified for the only sign that has not been described in literature as common to both these diseases seems to be the curious brownish discoloration of the skin and pigmentation of the cornea that were observed by Fleischer,[11] Völsch,[12] and Holloway.[13]

Stöcker[14] has recorded a case of progressive lenticular degeneration in which he found lesions in the lenticular nucleus which he believed to be characteristic of that condition, but, in addition, he found distinct alterations of the glia elements in all parts of the central nervous system except in the cerebellum, and he directs attention to the analogy that exists between progressive lenticular degeneration and the pseudosclerosis of Westphal and Strümpell.

On the other hand the cases of typical pseudosclerosis of Alzheimer and Hösslin[15] and A. Westphal[16] revealed not only the characteristic changes in the glia tissue in various parts of the brain, but these observers state that these alterations were most marked in the corpus striatum and the optic thalamus.

Considering both the clinical and the pathological evidence now at our disposal, even though this is somewhat limited, it would seem not at all unlikely that, in the near future, pseudosclerosis and progressive lenticular degeneration may be proved to be merely modifications of the one disease. At all events the cases of Stöcker, A. Westphal, and Hösslin and Alzheimer appear to justify the opinion that, on purely clinical grounds, a differentiation is not possible.

VALUE OF RECTAL FEEDING.[1]

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Everyone having experience with rectal feeding by the introduction of a pint or more of liquid nourishment at a time, is impressed with the discomfort and difficulties of continued nutrition by this means.

[10] Jour. Amer. Med. Assn., January 31, 1915.
[11] Deutsch. Ztschr. f. Nervenh., 1912, xl, 3.
[12] Loc. cit.
[13] Amer. Jour. Med. Sci., 1914, cxlviii, 235.
[14] Ztschr. f. d. ges. Neurol. u. Psychiat., 1914, xxv, 217.
[15] Ibid., 1912, viii.
[16] Arch. f. Psychiat., 1913, li.

[1] Read at a meeting of the American Gastroenterological Association, May 15, 1915.

An examination of stools shows how little food is really absorbed. On this account, for the past four years, I have been using the drop method as first introduced by Murphy for saline solution.

Four to eight ounces of peptonized milk with one pint of 4 per cent. sugar solution has been given three times daily. This method can be kept up for two weeks without discomfort. In a few cases they have caused cramp-like pains for the first day or two. In one or two cases vomiting has been produced. In but one case has it been necessary to give up the rectal feeding.

That the nourishment is carried well up to the intestines is shown by our experience in a case of fecal fistula after an appendix operation.

The patient was unable to retain any food by mouth, and rectal drop feeding was attempted. The dressings became invariably soaked with the enema after a few hours, showing that the fluid had made its way without difficulty through the length of the colon.

With rectal enemas of bismuth suspension there is no difficulty in obtaining a complete picture of the colon, but bismuth never enters the ileum except in the unusual cases of patent ileocecal valve.

With the drop method the same results were observed; no bismuth passed into the ileum.

In order to obtain some idea of absorption of rectal feedings by the Murphy method, metabolism investigations were made on cases of gastric and duodenal ulcer with no abnormality of nutrition. After the second day of artificial feeding, with nothing by mouth, the colonic intakes for four to six days in nitrogen was compared with the output in the urine and the return loss per rectum. Daily total nitrogen estimations of the urine were made while all the matter passed per rectum in the same period was collected, dried to constant weight, and the total estimated by the Kjeldahl method.

CASE I.

		Urine.		
Day.	Nitrogen intake.	Amount c.c.	Output T. N.	Stool.
3	1.95	11.25	11.82	0.78
4	1.95	7.00	9.79	0.78
5	1.95	13.80	11.72	0.78
6	1.95	10.10	12.19	0.78
7	1.95	16.00	12.12	0.78
	9.75		57.64	3.90
	Total output		3.90	
			61.50	
	Intake		9.75	
	Body loss in nitrogen		51.79	

CASE II.—Gastric ulcer.

Day.	Nitrogen intake.	Urine. Amount c.c.	Total nitrogen.	Amount from feeding.
3	1.95 gm.	10.75	12.82	1.39
4	1.95	9.05	12.01	1.39
5	1.95	8.00	11.15	1.39
6	1.95	8.25	10.20	1.39
7	1.95	8.50	9.63	1.39
	9.75 total nitrogen		66.81 urine 6.95 stool	6.95
			72.76 total nitrogen lost	
	Intake		9.75	
	Body loss in nitrogen		63.01 in five days	

CASE III.—Gastric ulcer (?).

Day.	Nitrogen intake.	Urine. Amount c.c.	Total nitrogen.	Amount from feeding.
3	1.95	9.80	10.09	0.91
4	1.95	8.40	9.42	0.91
5	1.95	9.20	9.61	0.91
6	1.95	8.70	8.72	0.91
	8.80		37.84 3.64	3.64
			41.48	
	Intake		8.80	
	Body loss in nitrogen		32.68 in four days.	

CASE IV.—Duodenal ulcer.

Day.	Nitrogen intake.	Urine. Amount c.c.	Total nitrogen.	Amount from feeding.
3	3.90	8.20	11.05	2.06
4	3.90	7.35	10.97	2.06
5	3.90	9.10	11.26	2.06
6	3.90	8.45	9.86	2.06
7	3.90	8.20	9.32	2.06
8	3.90	7.80	9.09	2.06
	23.40		71.55 12.36	12.36
			83.91	
	Intake		23.40	
	Body loss in nitrogen		60.51 in six days.	

CASE V.—Duodenal ulcer.

Day.	Nitrogen intake.	Urine. Amount c.c.	Total nitrogen.	Stools and amount from feeding.
3	2.92	8.80	11.56	2.19
4	2.92	7.50	9.80	2.19
5	2.92	7.60	10.68	2.19
6	2.92	8.60	10.39	2.19
7	2.92	7.60	9.28	2.19
		6.50	8.11	
	14.50			10.95
			59.82 10.95	
			70.77	
	Intake		14.50	
	Body loss		56.27 in five days.	

The average loss of total nitrogen by urine and stool in these five cases was 12.9 gm. per day. The average loss in nitrogen from metabolized proteid as shown by the total nitrogen of urine was 11.7 gm. per day.

The largest amount of nitrogen we were able to supply daily by rectal feeding was 3.9 gm., of which 2.06 gm. were returned with stools. According to the observations of Fr. Müller, the stools of individuals taking absolutely no nourishment contains 0.2 gm. per day. With this deduction from our estimations of the return nitrogen we have in no case observed an absorption of more than 50 per cent. of the proteid supplied, and it has fallen as low as 30 per cent. The average amount of nitrogen absorbed in these cases was 1.14 gm. per day.

When we compare the amount of nitrogen lost by these cases with the amount it was possible for them to absorb by colonic feeding, we realize how little we accomplish.

The difference between nutritional enteroclysis and normal salt enteroclysis was a little over one-tenth of the tissue albumin loss per day.

Over the periods of time that rectal feeding is usually carried out it is from practical standpoints an almost negligible quantity.

INTRACRANIAL TELANGIECTASIS: SYMPTOMATOLOGY AND TREATMENT, WITH REPORT OF TWO CASES.[1]

By Ernest Sachs, M.D.,

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Operations for Jacksonian epilepsy have so frequently resulted in negative findings that I desire to call attention to a group of cases which yields to treatment. This group, though apparently not large, has, nevertheless, a characteristic pathological picture. I shall briefly consider this condition from three points of view: (1) symptomatology; (2) pathology; (3) treatment. This paper is based on two typical cases.

Case I (Private record).—R. D., a boy, aged ten years, whom I saw in 1909, had, three months after a fall, fever and convulsions confined to the left side of the body, which began in the leg. A complete left-sided paralysis developed, which cleared up in a month. Following this the patient was apparently well and bright at school during the next year; then he began to have monthly convulsions, beginning in the face, with marked irritability; occasional headaches and vomiting; he often started suddenly. Wasser-

[1] Read at the meeting of the American Neurological Association, New York May 6 to 8, 1915.

mann negative. The physical examination revealed a left knee-jerk more active than the right; a double Babinski and normal eye-grounds; color fields could not be taken on account of the youth of the patient. On opening the dura (Fig. 1) a huge bluish mass of vessels protruded, which were so dense in places that the underlying cortex could not be seen. This mass lay over the motor area. There were no branches connecting with the dural vessels. I considered it inadvisable to try to remove this mass. Following the operation the patient had acute suppression of urine for twenty-

FIG. 1.—R. D., aged ten years. The very large size of the vessels of this telangiectasis is not sufficiently indicated on this drawing.

four hours, and then made an uneventful recovery. His condition remained unimproved. Therefore, at the family's urgent request, a month afterward I tried to remove this angiomatous mass, but the patient died twenty-four hours later. Autopsy showed grossly that the process was confined to the cortex. Unfortunately, no microscopic examination was made.

CASE II.—C. E., a boy, aged ten years, surgical number 1873. Admitted February 8, 1915, discharged February 23, 1915. First seen in November, 1914. Referred by Dr. Schwab and Dr. Wolfner.

His past history was unimportant. First convulsion when three months old. There have been five since, three of these in the past three months. Unconsciousness lasted from one to two hours. All convulsions begin in the wrist of the left hand. There are minor attacks, occurring as often as five or six times a day. In these the patient seems sleepy and has a numb feeling in his hand. Wassermann negative. Physical examination showed a weakness of the two lower branches of the left seventh nerve on emotional exertion, and a pale telangiectasis over the forehead. The left eye-ground

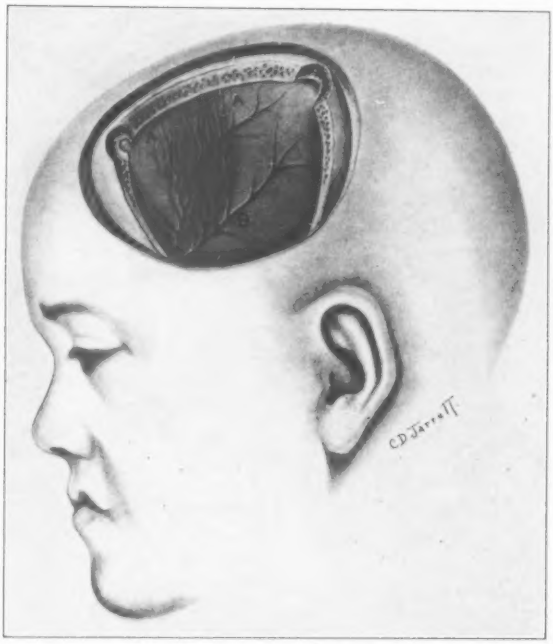

FIG. 2.—Case II.—C. E., aged ten years. Surgical No. 1873. Vessels were ligated at *A A* and *B B*.

showed an old choroidal atrophy; the right eye was normal. Again, the color fields could not be satisfactorily taken on account of the youth of the patient. An exploratory craniotomy was performed in February, 1915. An angiomatous process was found in the dura, which on reflecting the dura was found to have numerous connections with the pial vessels (Figs. 2 and 3). This area of increased vascularity lay over the lower part of the motor area. All the vessels connecting with the pial vessels were ligated and the dural mass ligated above and below; then the dura was closed. Two days after

the operation the patient had a violent convulsion. Since then he has been well. He has had three of the light attacks, of which he formerly had five to six daily. It is too early to express an opinion as to the ultimate result.

Both of these cases showed an unusual vascularity: the one of the vessels of the dura, which connected with the pial vessels, the other of the superficial vessels of the cortex.

In a careful survey of the literature I find that there really are very few cases like my second case. Virchow cites a case described

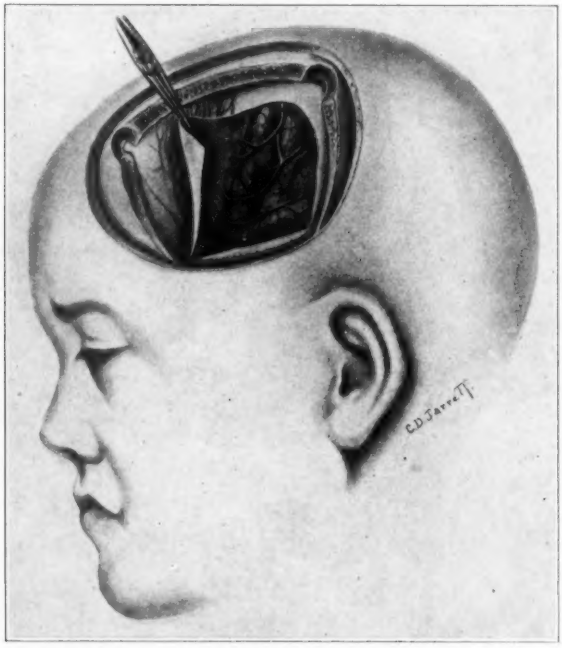

FIG. 3.—Case II.—The vessels shown running through the dura were all ligated.

by Hooper in his *Morbid Anatomy of the Human Brain*, published in 1828, which corresponds fairly closely. Pean gave a brief account before the French Academy of Medicine of an "angiome intracranien des meninges," which seems to have been identical.

Dr. Cushing, in 1906, described three cases of intracranial hemorrhage associated with trigeminal nevi. The process in all three of his cases was in the dura. He does not, however, mention the possibility that an identical process might occur on the cortex.

Oppenheim, in January, 1913, reported several similar cases in his paper on congenital brain tumors. He has given an admirable clinical picture of a condition which he calls angioma, which, in my opinion, is not an angioma at all but an entirely different pathological entity.

The first case is a type of which more have been observed. Krause in his book on the *Surgery of the Brain and Spinal Cord* reports two cases, one of which he has illustrated. This he calls an angioma venosum racemosum. In his opinion, evidently, he is dealing with a new growth—a true tumor. Tooth in his series of five hundred cases from the National Hospital has not a single instance of either condition. Bruns in his book on *Brain Tumors* makes the statement that cavernous angiomata and telangiectatic tumors have been found fairly frequently at operation,[2] and reports one case of what he calls an angioma which he observed himself. As he gives insufficient data it is impossible to decide to which of these two groups this case belonged. Virchow studied and described a number of such conditions, and was the first to clearly differentiate between telangiectases and true angiomata. He drew attention to the point which Adami emphasizes, namely, that a telangiectasis is a congenital dilatation of capillaries without any new-formed bloodvessels, while an angioma is a new growth and only properly so called when new bloodvessels are formed. In some of these cases the process is connected primarily with the capillaries, in some with the veins, and in others with the arteries. In true angiomata no vessels with all three coats developed are found, but in both of my cases the vessels looked prefectly normal, and were well formed.

In looking through the literature I find that these points are lost sight of and that the term angioma racemosum arteriale, or venosum, has been applied to conditions which were not actually new growths. I question very much whether Krause's case is correctly named. This, by the way, is the case cited by Oppenheim. It is the counterpart of my first case, and impresses me more like a telangiectatic process. Another point of interest is that cases like the two I have here reported are frequently, possibly always, associated with telangiectases elsewhere on the body, particularly on the face. My second case showed this. This was observed in Pean's case, also in Dr. Cushing's cases. Such cases as that of Emanuel, which he made the basis of an exhaustive discussion of angioma racemosus, are undoubtedly true new growths.

An etiological factor that has often been mentioned is trauma. Exactly what the relation of intracranial growths and trauma is, if any, is not at all clear. In certain cases it seems highly probable

[2] At the meeting at which this paper was read, several members reported on published cases.

that a trauma has either irritated a preëxisting new growth, making it develop rapidly, or has actually been the cause of the new growth. In my first case there was a definite history of this sort, but the operative findings, it seems to me, exclude that factor, since there was no evidence of inflammatory reaction such as adhesions, connective-tissue overgrowth, or roughened inner layer of the dura overlying the dilated vessels.

The classification of this process might seem merely of academic interest, but it has most important bearing on the treatment and prognosis. If this is a true new growth, extirpation is the procedure indicated, while if it is a telangiectasis, ligation should suffice. The question naturally arises, Will these thrombosed vessels, if left *in situ*, irritate the cortex, and should they therefore be removed after ligation? If possible the ligation should be planned so that the vessels do not remain filled with blood but are collapsed after the ligation. They will then atrophy and do no harm. This condition is a favorable one from an operative and prognostic stand-point.

It seems to me essential to distinguish more carefully between a true angioma—a neoplasm—and the process observed in these two cases, which is congenital and not a neoplasm. Undoubtedly some of the cases called angiomata have been misnamed. These cases, when reclassified, constitute a group which must be kept in mind in every case of Jacksonian epilepsy.

The clinical picture is quite a definite one.

1. Attacks of Jacksonian epilepsy occurring at long intervals in non-syphilitic young persons.
2. Unconsciousness of long duration.
3. No evidence of increased intracranial pressure.
4. A very slow progression of symptoms.
5. Telangiectases on the head or face.

For this condition I should like to suggest the name of intracranial telangiectasis.

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RESPIRATORY SIGNS AND SYMPTOMS IN TRICHINOSIS.

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One hundred and two case histories of trichinosis have been reviewed to determine the frequency of associated respiratory signs and symptoms which Dr. Edsall suggested may be more common

than is generally realized, and which may be so well marked as to cause difficulties in the diagnosis, particularly in the early stage of the disease before the typical eosinophilia develops.

It should be noted that rales are often found in the lungs, usually at the bases, in a variety of acute infectious diseases, depending somewhat on the height of the fever and prostration, and in this trichinosis proves to be no exception.

In the literature there is reference to abnormal respiratory signs and symptoms in trichinosis. However, all the authors quoted, as well as others, in relatively long articles, refer to these respiratory signs and symptoms only in a casual way, devoting but a sentence or short paragraph to them, except as a late or terminal complication of the disease. No attention is called to the fact that respiratory complications may obscure the true diagnosis.

Staubli[1] states that very early there develops a bronchial catarrh. He explains that this is dependent upon the invasion by trichinellæ of the respiratory muscles, bringing about their insufficiency which leads to a scanty expectoration and ultimately to bronchopneumonic processes. Staubli remarks that the sudden rises of temperature with chills may be dependent on this.

Besides the mechanical involvement and irritation of the respiratory muscles by the embryos there is good evidence from the fact that the embryos occur in the blood stream and great veins (Staubli,[2] Herrick and Janeway,[3] Lamb,[4] Cross[5]) to show that they may be present in the lung tissue and cause irritation of the alveoli and bronchial mucosa directly.

Thompson,[6] in a report of 52 cases, says "cough may be present in a number of cases with a slight bronchitis, probably due to irritation of the bronchi by the parasites, but the latter were not found in any of the sputa examined."

Kratz[7] reported a total of 337 cases. Sixty-four of his 101 deaths were attributed to disturbances of the respiratory apparatus and 14 of these had hypostatic pneumonia. Rupprecht[8] also mentions pneumonia as a complication, especially in fatal cases.

Stiles[9] in his description says: "The respiration becomes difficult and respiratory troubles are likely to be severe, especially in the fourth and fifth week, and there may be severe dyspnea accompanied by violent asthma." Granger[10] also expresses the same view, though in his 18 cases reported no mention is made of any such

[1] J. F. Bergmann, Wiesbaden, 1909.
[2] Loc. cit.
[3] Arch. Int. Med., 1909, iii, 203.
[4] Amer. Jour. Med. Sci., 1910, cxlii, 395.
[5] Arch. Int. Med., 1910, vi, 301.
[6] Amer. Jour. Med. Sci., 1910, cxl, 157.
[7] Die Trichinenepidemie zu Hedersleben, Leipzig, 1886; Berl. klin. Wchnschr., 1865, lii, 509 (quoted by Staubli).
[8] Die Trichinenkrankheiten im Spiegel der Hettstädter Endemie betrachtet Hettstadt, 1864 (quoted by Staubli).
[9] Osler's Modern Medicine, 1907, i, 605.
[10] St. Paul Med. Jour., 1914, xvi, 399.

respiratory signs or symptoms. Steiner[11] mentions involvement of the respiratory muscles by the parasites and the resulting paralysis as a cause of death.

Mosler and Peiper[12] found bronchial catarrh only rarely. They do not mention pneumonia; while Leen[13] speaks of bronchial catarrh, which may at times amount to a bronchopneumonia or lobar pneumonia, but with no reference as to what stage of the disease this occurs or with what frequency.

Staubli says that at autopsy in a large majority of cases Cohnheim found a marked symmetrical bronchitis with frequent hypostatic conditions in one or both lungs but emphasizes that embolic processes are never seen. Frothingham[14] found hemorrhages into the lung tissue with embryos present, as did Askanazy,[15] who mentions embryos in the lung alveoli. They have never, however, been found in the sputum.

Romanovitch's[16] research has demonstrated anew that the trichina sows microbes along its path as it passes from the intestinal mucosa to the thoracic duct, great veins, and general circulation, and hence that the dominant character of the infection due to trichinæ is polymicrobian. He quotes Friedrich, who reports finding subcutaneous abscesses containing the trichinella, and he himself obtained positive cultures in 13 out of 23 rats killed at various intervals after infection with trichinæ and in 7 out of 10 guinea-pigs. He therefore concludes that fever, abscesses, and the fatal septicemia observed in man are due to microbes inoculated by the trichinæ. This offers an explanation of why lung infections may be common to this disease.

The 102 cases of trichinosis that are reviewed here have been taken from the records of the Massachusetts General Hospital (1899 to 1914), the Presbyterian Hospital in New York (1906 to 1914), and the Johns Hopkins Hospital (1899 to 1914).

The diagnosis in 70 per cent. of these cases was made by finding the embryo in excised pieces of muscle or in the blood; in 5 per cent. no parasite was found, but the muscle showed a histological picture of a myositis such as is found when trichinæ are present; in 25 per cent. by the history, symptoms, and physical signs, and the eosinophilia which varied from 13 to 63 per cent. of the total leukocyte count.

Those cases in which the final diagnosis of trichinosis was in doubt or in which a chronic cardiac or pulmonary condition existed have been excluded. One case that died was excluded because of

[11] Boston Med. and Surg. Jour., 1908, cli, 721.

[12] Tiersche Parasiten in Nothnagel's spez. Patholog. u. Therap., 1894, vol. vi (quoted by Staubli).

[13] Boston Med. and Surg. Jour., 1913, clxviii, 601.

[14] Jour. Med. Research, 1906, xv, 487.

[15] Virchows Arch., 1895, cxli, 42 (quoted by Staubli).

[16] Comp.-rend. Soc. de biol., 1911, lxx, 257 and 339.

an incomplete record. All of the 102 cases recovered except one, and it should be noted that although this patient had moderate dyspnea, no abnormal physical signs in the lungs were detected.

Five of these cases are of particular interest, because of respiratory complications, and are described in some detail later, leaving 97 cases which are here summarized; 8.8 per cent. of the total 102 cases (4 of the 5 cases described later and 5 of the 97 cases summarized) showed respiratory signs and symptoms severe enough to lead to the serious consideration or actual diagnosis of a purely pulmonary condition in the preliminary diagnosis.

Thoracic pain, usually in the region of the diaphragm or along the lower intercostal muscles, was not an uncommon symptom, but bore no relation to the presence of a slight or severe bronchitis, though usually when present was associated with a mild dyspnea and often with a shallow type of respiration.

The rate of respiration was usually increased with the fever to 25 to 32 per minute, reaching as high as 46, again varying with no regularity in regard to physical signs in the lungs except for the fact that those cases where the bronchitis was most marked usually had a higher rate than the others.

Of the 97 cases, 51 (50 per cent. of the total 102 cases) had no cough, nor was there any abnormality of the lungs noted in the physical examination during the first few days in the hospital or subsequently. The 46 remaining cases may be divided into three groups as follows:

I. Fifteen cases (15.3 per cent. of the 97) had cough without abnormal pulmonary physical signs, and only 3 of these raised any sputum.

II. Thirteen cases (13.6 per cent.) had not only a cough, but also abnormal physical signs were present in the lungs, though only 7 of them raised sputum. Thus 28 cases or 29 per cent. had a cough which began one to twelve days, usually about two to six days, after the earliest symptom of the disease, lasting three to twenty-five days, or through the course of the illness.

III. Eighteen cases (18.5 per cent.) had pathological physical signs of the lungs without there being reference to cough in the records.

Thirty-one cases, or 31.9 per cent. of the 97 cases (all the cases of groups II and III), showed abnormal physical signs in the lungs, which may be described as "very slight," "definite," or "well marked," according to the number of rales present.

(*a*) Very slight signs were noted in 11 cases, and consisted in 4 cases of rare dry rales, which are described in 2 cases as being scattered and in the other 2 as being at the bases. In 7 cases they consisted of a "few moist rales." In 3 of the 7 they were scattered, and in 4 were at the angles of the scapulæ.

(*b*) Definite signs, that is, distinctly more numerous rales, were found in 10 cases. In 1 they were "scattered subcrepitant" and

in the 9 others moist (fine, medium, or coarse), located at the base in 4, the right axilla in 1, both apices in 1, the angles of the scapulæ in 1, and scattered in 2.

(*c*) Well-marked signs (rales very plentiful) were noted in 10 cases. In 2 of these the rales were of a dry type, in 1 at the bases only, and in the other "scattered all over." In the other 8 cases the rales were moist and of all varieties. In 5 of these they were everywhere in the lungs. In 2 at both bases, and in 1 at the left base only.

Ten of these 31 cases which had rales showed additional abnormal physical signs as follows:

Four had slight dulness with diminished respiration when the rales were at the bases.

At the point where the rales were most numerous, 2 showed slightly harsher breathing than normal, while 3 were noted to have suppression of breath sounds.

One case with rales at the left base showed dulness in this region and in the left axilla, where the breathing was distinctly diminished, but Roentgen-ray examination revealed only "peribronchial thickening."

The physical signs were first noted within forty-eight hours after the patient entered the hospital in 29 cases, while in the 2 others they developed within six days after admission, about eight days after the onset of the disease.

It has been difficult to tell from the records the exact duration of the abnormal signs in the lungs, though as long as the temperature remained elevated the signs persisted. However, it seems that as the temperature fell the signs began to be less and disappeared when it became normal, though in a few cases lasting some days longer. In the cases with the mildest signs they occurred only at the height of the fever and disappeared several days before the temperature was normal.

There was reference to sputum in 13 cases of the 102. No mention was made in the record of cough or sputum in one of these cases, yet a laboratory examination of sputum was noted.

In 7 cases the macroscopic examination of the sputum alone was noted; 3 were blood-tinged, of mucopurulent nature; 2 were "grayish white," while 2 were "mucopurulent."

Microscopic examination was made of 6 sputa, 4 of which grossly were "mucopurulent." At one time one of these showed blood streaks and 2 were "white mucoid."

Smears showed no tubercle bacilli in any of the sputa; many influenza bacilli were seen in 1 besides other organisms; 4 others showed a mixed growth of bacteria. The case which showed at first blood streaks in a mucopurulent sputum and later was only mucopurulent, contained many influenza bacilli. No eosinophile cells, Charcot-Leyden crystals, or parasites were discovered.

The following 5 cases, not included in the 97 cases referred to above, serve to show how pulmonary complications may obscure the diagnosis of trichinosis.

CASE I.—This illustrates how a bronchitis or mild bronchopneumonia may be a prominent feature from the onset of the disease associated with a delayed eosinophilia.

Male, negro, aged thirty-four years, works in tunnels and is subject to decompression and compression for five minutes many times a day. *Family history, past history,* and *habits,* unimportant.

Present Illness. For three weeks he has had a cough, increasing in intensity, raising yellowish-white sputum which in the past two days has contained a little blood. During this time he has had a persistent soreness and tenderness of his legs, though not of a severe nature. Night sweats have been frequent.

For two weeks he has had a severe temporal headache, and has felt feverish, and during the past week his eyelids have been a little puffy in the morning.

Physical Examination. A well-developed negro, looking sick and coughing, having moderate tenderness of the leg muscles.

Nodes. Generally palpable. Rest of examination negative except for heart and lungs.

Heart. Normal size; sounds regular; harsh, short, systolic murmur is heard at the right second interspace, transmitted to clavicular region but not to neck. Second sounds are normal.

Lungs. Over both bases and in both axillæ, extending higher on left, are heard many fine, medium, and coarse moist rales. At a point half-way between the base of the left lung and angle of the scapula is an area of dulness where there is distant breathing of higher pitch than normal, with slightly prolonged expiration associated with a distant bronchial whispered voice.

Day in hospital.	Temperature.	Pulse average.	Respiration.
1st to 6th . . .	101.0° to 104.0°	100	30 to 25
6th to 16th . . .	98.0° to 100.0°	80	25 to 20
16th to 21st . . .	98.6° to 101.0°	85	25 to 20
21st to 33d . . .	98.0° to 99.5°	75	20

Blood: Hemaglobin, 90 per cent.

Day in hospital.	White count.	Polynuclear neutrophiles and transitionals.	Lymphocytes.	Eosinophiles.	Mast cells.
1st . .	10,500	86 per cent.	12 per cent.	2 per cent.	0 per cent.
5th . .	. . .	88 "	10 "	4 "	0 "
10th . .	9,300	80 "	8 "	10 "	2 "
17th . .	. . .	69 "	13 "	18 "	0 "
27th . .	. . .	60 "	25 "	15 "	0 "

Trichinæ were found in a piece of gastrocnemius muscle on the sixth day after admission.

Sputum. On entrance this was "frothy-mucoid," with some purulent particles mixed with blood. A few influenza bacilli were

present, otherwise negative. On the third to sixth day in the hospital, several examinations showed a mucopurulent sputum with many influenza bacilli. On the nineteenth day in the hospital a thick mucopurulent sputum was expectorated.

The signs present in the lungs persisted for five days, then gradually disappeared by the twelfth day. On the sixteenth day after entrance the moist rales reappeared, and on the twentieth day the area of dulness at the left base with harsh inspiration and expiration reappeared. A Roentgen-ray taken on the twenty-second day showed "the heart slightly enlarged to the right; question of dilated aortic arch. No tuberculosis." After this the lungs cleared and were quite free of rales on the twenty-ninth day.

CASE II.—This case, though exhibiting no signs in the lungs, had an onset of a respiratory nature which caused the physicians to believe for some days they were dealing with a purely thoracic disease.

Male, white, aged twenty-six years; occupation, clerk.

Family history and *habits* unimportant.

Past history negative except for pneumonia three years ago.

Present Illness. Nine days ago he began to have a severe "cold," feeling very chilly, mean and weak since then. He has had a cough associated with slight pain in the front of the thorax, raising very little sputum; constant throbbing headache, aggravated by cough, has been persistent. There were no other symptoms.

Physical Examination. Negative throughout, except for neglected teeth with sordes and a few herpetic lesions on the lips. Temperature varied from 102° to 103°, gradually reaching normal on the eighteenth day after admission. The respiration was 25 to 30 per minute on the day of entrance. The day after 32 to 35, falling to 24 to 22 on the eighth day, and then averaging 20. The pulse during the fever was 100 to 90.

White count on entrance 11,800. (No differential count was made.)

Three days after entrance a few more herpetic vesicles developed on the lips. The record states the lungs were still negative, and the patient continues to cough a little. On this day a blood smear showed 7 per cent. eosinophiles.

Four days after entrance tenderness of the calves of the legs with pains "all over" developed.

On the eighth day marked muscle symptoms were present and a piece of excised muscle revealed trichinæ.

On the ninth day cough was still present, but with very little sputum. The symptoms gradually disappeared as the temperature dropped, the cough going by the eighteenth day in the hospital, when the temperature reached normal.

CASE III.—The following case illustrates an onset with respiratory symptoms. A latent tuberculosis, possibly lighted up by the

trichinosis, cannot be ruled out. It does not seem as if all the physical signs in the lungs and the respiratory symptoms could have been due to a previously quiescent tuberculosis, but that the trichinosis, which had existed for some time, was associated with a bronchitis.

Girl, aged thirteen years.

Family history and *past history* negative.

Present Illness. Three weeks ago she "caught cold" and had a chill that night; since then she has coughed a good deal, raising small amounts of yellowish-white sputum; breathing at times has lately caused slight pains in her chest. She has felt feverish, had severe headache, and sweats frequently at night. Pains in the legs and thighs have occurred, but were not severe.

Physical Examination. Very slight puffiness of eyes and tenderness on pressure of calves of legs and lower ribs was noted on examination, which was otherwise negative except for the lungs.

Lungs. Percussion showed the note in the left infraclavicular region slightly higher pitched than on the right. In the back a doubtful slight impairment in resonance at the left apex and at both bases. Breath sounds over upper left front rougher than normal, with expiration prolonged. Fremitus normal. Over both backs were numerous fine moist rales and at the bases sonorous rales; a question of an occasional dry crackling rale at the left infraclavicular region was noted.

Day in hospital.	Temperature.	Pulse average.	Respiration.
1st to 5th . .	101.0° to 99.5°	100	20 to 24
5th to 10th . .	98.0° to 99.0°	80 to 70	20
10th to 30th . .	96.5° to 97.8°	80 to 60	20

Blood.	White count.	Eosinophiles.
On entrance	24,840	53 per cent.
Twenty-seven days later	17,120	56 "

Calmette tuberculin conjunctival test, 1 per cent. negative; Calmette tuberculin conjunctival test, 5 per cent. positive.

Stools, urine, and blood culture negative.

A very small piece of muscle excised two days after entrance showed "no trichinæ."

The moist rales in the lungs were not heard after a few days, nor were the dry ones noted after the first examination. The slight dulness below the left clavicle with roughened breath sounds persisted. Neither cough nor sputum was mentioned in the record after the first three days in the ward. The tenderness of the calves of the legs and lower ribs persisted for the first week, and was not severe.

CASE IV.—The case illustrates a sudden onset resembling pneumonia.

Male, aged twenty-three years, clerk in a grocery store.

Family history, past history, and *habits* unimportant.

Present Illness. Twenty-four hours before admission he was seized with a sharp pain in the left side of his chest, increased by breathing and coughing. The pain shifted to the right axilla and became more severe during the night. There has been some cough without expectoration. He had a chill this morning and one just after admission. He has felt feverish and prostrated since the onset.

Physical Examination. A well-developed and nourished man, appearing acutely ill, lying quietly in bed. Temperature, 103; pulse, 108; respirations, 30. The examination was negative except for much mucus in the throat, and the lungs showed some dulness in the right upper axilla, with slight whiffy bronchial expiration, but nothing definite.

Day in hospital.	Temperature.	Pulse.	Respiration.
1st to 6th . .	104.0° to 100.0°	110 to 78	30 to 20
6th to 12th . .	101.0° to 99.0°	100 to 75	24 to 20
12th to 17th . .	98.2° to 99.2°	88 to 74	24 to 20

The Roentgen-ray, upon the day of entrance showed "throughout greater portion of right lung shadow there is slight increase in density, and there are patches which strongly suggest early tuberculosis—slight irregularity in diaphragm shadow."

Five days after entrance all tendon reflexes were absent except the right knee-jerk and both triceps, which gave but a feeble response; and the note reads, "The signs in the lungs become no clearer; dulness on right side slight, but persists; breathing thought to be increased in apex of right axilla and interscapular region." The patient was quite cyanotic at this time. The record makes no further mention of the lungs or gives clinical notes of importance. No rales were ever present. The patient was discharged "well" sixteen days after entrance.

	Blood, white count.	Polynuclear neutrophiles.	Lymphocytes.	Eosinophiles.	Mast cells.
On admission . .	11,000	67 per cent.	23 per cent.	8 per cent.	2 per cent.
Six days later . .	. .	72 "	19 "	9 "	0 "

Reds, 4,950,000; hemoglobin, 90 per cent.

The urine, stool, Wassermann, von Pirquet, and blood cultures were all negative.

Muscle excised nine days after entrance showed a distinct myositis with wandering cells and eosinophiles, and the muscle fibers show various stages of degeneration. Diagnosis: "myositis due to trichinæ."

CASE V.—This case resembles pneumonia. Unfortunately satisfactory history was not obtained, owing to his nationality; and a specimen of muscle was unobtainable. The diagnosis here is based on the eosinophilia.

Male, aged forty-four years, Italian laborer.

Family and *past history* unrecorded.

Present Illness. For two weeks he has felt sick and had a bad cough. During the past week he has been in bed with increasingly severe cough, chilly sensations, constant headache, and pains all over his body, especially in his legs.

Physical Examination. Well-developed man looking acutely ill and anxious, breathing rapidly, forty to the minute. The examination was entirely negative except for pyorrhea, sordes of teeth, and a heavily coated tongue. The temperature ran irregularly 101° to 103° in the morning, falling to 100° to 99° in the afternoon for the first ten days after admission, gradually declining after the first eight days. The following eleven days the temperature was normal. The pulse averaged 82 during the pyrexia and 74 when the temperature was normal. The respirations ran from 28 to 34 in the morning during the first eight days, except that in the first two days they were 44; in the evening they ran from 22 to 18 (perhaps due to morphin). With the descent of the temperature the respiration returned to normal.

Three days after admission a few moist rales without other signs were noted in the lower right axilla at the bases. The spleen was just palpable, otherwise examination remained negative. Nine days after admission a note states that "the patient continues to have a hacking cough, raising a little yellowish material." The lungs showed some scattered rales at the bases, present the next day also, though it is not stated when they cleared.

The patient was discharged twenty-two days after admission, having shown no abnormal symptoms or signs after the sixteenth day. No reference is found in the record of positive or negative evidence of muscle tenderness or other symptoms of trichinosis.

Day in hospital.	Blood, white count.	Polynuclear neutrophiles and transitionals.	Lymphocytes and large mononuclears.	Eosinophiles.
2nd	25,600	91 per cent.	9.0 per cent.	0.0 per cent.
10th	26,600	22 "	9.5 "	68.5 "
12th	27,000	26 "	11.0 "	63.0 "

Blood cultures, Widals for the typhoid group, sputum, urine, and feces were all negative.

From the above group summaries of the histories and physical signs in these cases it may not appear evident that there was sufficient reason for being misled at first as to the diagnosis. It is evident, however, from the complete records that such was the case. In the two cases that we personally had the opportunity to see and in another in regard to which we talked with the physician in charge, such certainly was the case, the pulmonary symptoms entirely obscuring the more ordinary ones of trichinosis for a time. It is quite probable that but for the hospital routine blood examina-

tion the proper diagnosis would never have been reached in some of the cases.

In Case I, which was seen by both of us, though the record states that the patient had muscle tenderness, it was not at all prominent when compared to the picture of an evidently sick man persistently coughing and whose lungs were filled with rales. Later when 4 per cent. of eosinophiles were found the muscle tenderness was given more consideration, which led to the finding of trichinæ in the excised muscle. For the first few days certainly the lung symptoms obscured everything else.

Case II had a history not unlike pneumonia: the patient looked sick, had a high temperature, rapid respiration, and labial herpes. It was not until the eosinophilia and muscle symptoms developed that trichinosis was considered.

In Case III trichinosis was made the probable diagnosis at entrance, owing to the high percentage of eosinophiles; yet the history given by the patient and her family was of purely respiratory character, and she exhibited well-marked signs of a bronchitis. The pains in the legs and edema of the eyes were both slight and noted only after finding the eosinophilia.

Those who saw Case IV felt at first they were dealing with a pneumonia, owing to the acute onset of symptoms of respiratory nature, with fever and signs in the lungs. Trichinosis did not occur to them until the second differential count of the white cells was made. The pulmonary signs might be explained by an old tuberculosis.

Again, Case V resembled pneumonia. The history was purely respiratory, the respiration high, and rales were present in the lungs. Trichinosis was not considered until the high eosinophilia was found on the tenth day after entrance.

Conclusions. In the literature, abnormal respiratory signs and symptoms in trichinosis have been noted, but without emphasis on the fact that they may obscure the diagnosis, especially when the patient is first seen. Bronchitis, bronchopneumonia, and lobar pneumonia have been spoken of, especially as a terminal event and chiefly in the earlier reported cases in Germany.

In 50 per cent. of the 102 cases studied there is no mention of cough or abnormal lung signs at any time during the course of the disease, but in the remaining 50 per cent., 16 cases (15.6 per cent.),[17] had cough without abnormal physical signs in the lungs, while 17 (16.6 per cent.) had cough with abnormal lung signs, making 33 cases (32.3 per cent.) that had cough beginning one to twelve days (usually two to six) after the onset of the disease and lasting three to twenty-five days or throughout the course of the illness; 18 cases (17.6 per cent.) had abnormal signs in the lungs but without

[17] This and the percentages given below are percentages of the total 102 cases.

cough, making 35 cases (34.5 per cent.) that had abnormal physical signs in the lungs. These physical signs were present within forty-eight hours after the patient entered the hospital in 32 cases (31.5 per cent.), and in 3 cases (2.9 per cent.) within six days. The duration of these signs seemed to depend on the length of time the temperature remained elevated, the signs disappearing as the temperature fell. If the signs were slight, they existed, as a rule, but for a few days, and only at the height of the fever.

Nine cases (8.8 per cent.) showed respiratory signs or symptoms, or both, severe enough to lead to a serious consideration or actual diagnosis of a purely pulmonary condition during the first few days the patients were in the hospital.

This percentage of cases leading to error in the diagnosis is greater than is usually recognized.

We acknowledge our indebtedness to Dr. Janeway, Dr. Longcope, and Dr. Edsall for permission to use their hospital records.

A TEST OF MYOCARDIAL EFFICIENCY, WITH REVIEW OF ONE HUNDRED AND TWENTY-SEVEN CASES.

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Some two or three years ago my attention was called to the fact that variation in systolic blood-pressure in relation to posture and exercise might be interpreted in terms of myocardial efficiency. Since then I have used a certain uniform method in a sufficient number of cases to warrant a review. Not all cases were studied in this manner, nor were any studied with the intention of tabulation. From the beginning the method has grown in favor, and I have come to look upon the blood-pressure picture—that is, the relation to each other of pulse, systolic, diastolic, and pulse pressure taken with the patient reclining and fully relaxed and again standing after light exercise—as a fair indication of myocardial efficiency. In these determinations I uniformly take the pulse, systolic blood-pressure, and diastolic blood-pressure with the patient reclining and fully relaxed; then I have him walk at his ordinary speed four times the length of my office, about 75 feet, and repeat the test in the standing position. This exertion is not sufficient to cause such subjective symptoms as dyspnea, palpitation, or chest pain unless the heart muscle be much impaired. It should not do this. It is a postural test plus sufficient exertion only to mildly stimulate the cardiovascular system.

I have tabulated all cases recorded, grouping them according to myocardial efficiency. It is obvious that a grouping on a basis of myocardial efficiency is open to error, and at the best can only be relative. I believe, however, that the lines of classification are sufficiently definite for the purpose.

Four groups are employed, namely, "good," "fair," "poor," and "bad." In group "good" are only normal or functionally normal hearts. In group "bad" are only those with broken (lost) compensation. It so happens that in the classification none appear in this group for the reason that the test was applied only to walking cases. The intermediate groups, "fair" and "poor," are not easily defined. In general, however, the cases in "fair" displayed mild subjective symptoms, while in "poor" the subjective symptoms were pronounced. I have subdivided group "fair" into "fair," "fair plus," and "fair minus" for reasons which appear in the text. In tabulating, in order to avoid the easy pitfall of making the case fit the conclusion, I first grouped the case and then noted the pulse, systolic and diastolic pressure and the pulse pressure reclining, and standing after light exercise. The grouping was governed by the subjective symptoms and physical signs as recorded and by my personal knowledge of the cases. The cases being selected from the heart clinic of a sanitarium, naturally the largest number fall in group "fair," for the reason that in such a clinic, walking cases predominate.

In Table I, not published, I have grouped the cases according to myocardial efficiency, that is, "good," "fair," or "poor," and have recorded pulse, systolic pressure, diastolic pressure, and pulse pressure, patient reclining and fully relaxed, and again standing after light exercise, together with the change in systolic pressure and pulse pressure, and the diagnosis.

TABLE II.

Group.	Whole number.	Increased or stationary S. B. P.	Per cent.	Decreased systolic blood-pressure.	Per cent.	Increased pulse pressure.	Per cent.	Stationary pulse pressure.	Per cent.	Decreased pulse pressure.	Per cent.
"Good"	13	13	100	0	0	11	85	2	15	0	0
"Fair"	85	55	65	30	35	35	41	24	28	26	31
"Poor"	29	12	41	17	59	6	21	6	21	17	58
Subgroup.											
Fair+	17	..	77	..	23	..	53	..	29	..	18
Fair−	11	..	9	..	91	..	18	..	36	..	45

Table II is a summary giving under each myocardial efficiency group the whole number of cases appearing in that group, the number with increased or stationary systolic pressure, the number with decreased systolic pressure, and the number with increased stationary or decreased pulse pressure, each being noted also in

terms of percentage. Table II best shows the contrasts and is the easiest comprehended if we read alone the percentage columns. For instance, in group "good" there are thirteen cases. In 100 per cent. there was increased or stationary systolic pressure; in 85 per cent. there was increased pulse pressure. In 15 per cent. stationary pulse pressure. In none, decreased pulse pressure. Group "fair" 85 cases, in 65 per cent. increased or stationary systolic pressure, in 35 per cent. decreased systolic pressure; in 41 per cent. increased pulse pressure, in 28 per cent. stationary pulse pressure, in 31 per cent. decreased pulse pressure.

Group "poor," 29 cases. In 41 per cent. there was increased or stationary systolic pressure; in 59 per cent. decreased systolic pressure; in 21 per cent. there was increased pulse pressure, in 21 per cent. stationary pulse pressure, in 58 per cent. decreased pulse pressure.

In the subdivision, for the purpose of emphasizing the main point, I have noted in terms of percentage only, cases in "fair plus" (+) and "fair minus" (−). It is to be remembered that "fair plus" signifies bordering on good, and "fair minus" signifies bordering on poor.

In the 17 cases recorded as "fair plus," there was increased or stationary systolic pressure in 77 per cent., decreased in 23 per cent., increased pulse pressure in 53 per cent., stationary pulse pressure in 29 per cent., decreased pulse pressure in 18 per cent. In the 11 cases recorded as "fair minus" there was increased or stationary systolic pressure in 9 per cent., decreased in 91 per cent., increased pulse pressure in 18 per cent., stationary pulse pressure in 36 per cent., decreased pulse pressure in 45 per cent.

Conclusions. In the estimation of myocardial efficiency, systolic blood-pressure and pulse-pressure variations together with changes in pulse rate in relation to postures and light exercise have positive value.

Under the test described an increase in systolic pressure together with a stationary or increased pulse pressure is suggestive of fairly good myocardial efficiency.

A decrease in systolic pressure together with a decrease in pulse pressure is suggestive of poor to bad myocardial efficiency.

While the cases reported do not furnish data for comparative study in the individual case, it is obvious that repeated applications of the test, under uniform conditions, should furnish in the individual case, evidence in relation to progress.

REVIEWS

COLLECTED PAPERS OF THE MAYO CLINIC, ROCHESTER, MINNESOTA. Edited by MRS. M. H. MELLISH. Vol. VI, 1914. Pp. 814; 347 illustrations. Philadelphia and London; W. B. Saunders Company, 1915.

THIS the sixth volume of these collected papers is the first of the series to be numbered, and appears with a slightly changed title, no reference now being made to St. Mary's Hospital. This no doubt is to be explained by a wish to give public recognition to the fact that a fair proportion of the work here represented is done outside of St. Mary's Hospital, and by some who have no direct affiliation with it. Though this hospital is still the centre of the clinical work of the Mayo Clinic, there are several other hospitals or *sanatoria* in Rochester, in addition to the large clinic building—all of them quite distinct from St. Mary's Hospital, yet a part of the "Mayo Clinic."

A thought that has impressed itself in reviewing this volume, as it has in reviewing its predecessors, is this: Why is not the official status of each of the contributors to the volumes defined somewhere? It is undeniable that a comparatively unknown name carries more weight if there is some indication of the amount of personal experience which the writer possesses. For example: in the present volume there is a paper on the use of the Roentgen rays in the diagnosis of pulmonary tuberculosis; it is stated that it is based on the combined radiographic and clinical findings in a series of 1000 cases of pulmonary tuberculosis, but the writer's status in the Mayo Clinic is not indicated, and from the context cannot be inferred. Some of the other contributors refer glibly to the three, four, or ten or more thousand cases of a certain affection observed in "our clinic," when it is quite evident that they refer not to cases personally studied, but to the entire material of the Mayo Clinic since its origin. In the same sense just consider the hundreds of thousands of cases to which a newcomer could refer if writing as a representative of the Hôtel Dieu at Paris or the Allgemeine Krankenhaus at Vienna.

When one comes to look back over the work and progress of the Mayo Clinic, it seems that three more or less distinct periods can be recognized, which are an epitome of the development of the surgical

mind. The earliest period may be considered one characterized by descriptions of technique. In few other clinics in the world have surgeons so thoroughly systematized the mechanical details of the operations in common use in abdominal surgery, particularly in the surgery of the stomach and duodenum. And no one who has given thought to the matter fails to recognize that the highest merit of any operation is that it shall be reduced to a type and become a mere mechanical procedure in which one step follows the other with clock-work regularity, without let or hindrance. When this has been accomplished and the mechanical details have become familiar to surgeon and onlookers alike, then the operation as an operation has lost its interest.

The second period of development, and that which naturally succeeds the merely technical period, is the period during which the immediate mortality and the end-results of operations are of paramount interest. This period lasted a long time in the Mayo Clinic; and though the questions arising from the immediate mortality have nearly all been answered, those relating to the ultimate results are not entirely settled.

The final stage of development is that during which interest centres in pathology—the causes and natural course of disease; and it is this period on which the Mayo Clinic is now entering. The present volume, for instance, contains a great deal of matter relating to the pathogenesis of gastric carcinoma, and though it is rumored that some pathologists in other parts of the country do not think the conclusions reached by those they consider the "amateur" pathologists of the Mayo Clinic are warranted by the facts presented, it is of value, nevertheless, to have aroused discussion, and to have made the professional pathologists see the necessity for giving reasons for the faith or lack of faith that is in them.

One word more may be permitted the reviewer in closing. He knows that comparisons are odious, yet a comparison between the two chief surgical clinics of the country in a measure imposes itself. There is another clinic in this country—that at the Lakeside Hospital, Cleveland, where the main interest in the clinic from the very beginning of its career has centred in questions of pathology and pathological physiology. The technique of operation has never been neglected, for nowhere else in the world, we firmly believe, are operative details so accurately and faultlessly accomplished; and the immediate and ultimate results of treatment are nowhere else so studiously observed nor so excellent; but both technique and mortality statistics have always been subordinated to the overwhelming interest in the pathology of disease as it presents itself to the surgeon. The Mayo Clinic has achieved Roman citizenship at the price of fifteen years of hard toil; but the Lakeside Clinic was free born. A. P. C. A.

DISEASES OF THE SKIN. By JAMES H. SEQUEIRA, M.D. (Lond.), F.R.C.P. (Lond.), F.R.C.S. (Eng.), Physician to the Skin Department and Lecturer at the London Hospital; in charge of Queen Alexandra's Department for Light Treatment; Consulting Dermatologist to the Radium Institute; Secretary of the British Section of the International Association of Dermatology and Syphilis, etc. Second edition; pp. 650; 48 plates in colors and 238 text figures. Philadelphia: P. Blakiston's Son & Co., 1915.

THE author has made a careful revision of this attractive book and has added one hundred and eleven pages, four new plates in color, and sixty photographs in the text. The work has been made of greater value to the investigator by the addition of several photomicrographs. Ringworm has been brought scientifically forward by plates showing the characteristics of various cultures. The writer treats of his subject by chapter headings, insofar as it is possible, from the etiological point of view. Eczema and also syphilis are treated upon in separate chapters, rather than in the former edition, with other diseases. Urticaria pigmentosa has been removed from "tumors of the skin" and placed under "toxic eruptions." Piedra (trichosporosis) has been transferred from "diseases of the appendages" to "vegetable parasitic affections." A considerable number of conditions have been added, such as grain itch, leukemia cutis, larva migrans, eczematoid ringworm, tropical ringworm, recurrent erysipelatoid eruptions on the face, trichomycosis axillaris flava, and rubra et nigra.

There has been a considerable amplification of pellagra, sporotrichosis, carcinoma, and epithelioma. Syphilis has been brought up to date by the description of Noguchi's luetin reaction, the exact method of giving salvarsan, and its contra-indications.

Although the book is not as large as several other text-books on dermatology, the large number of photographs in colors, most of which are excellent, make the volume a valuable asset to those interested in cutaneous medicine. F. C. K.

BLACK'S MEDICAL DICTIONARY. By JOHN D. COMRIE, M.A., B.Sc., M.D., F.R.C.P. (Edin.). Fifth edition; pp. 858; 431 illustrations, 12 plates. New York: The Macmillan Company. London: Adam & Charles Black, 1914.

THE appearance of a fifth edition speaks well for the popularity of this book, but it is important for the medical man especially to know just what its scope is. The "original intention," which

"has been sedulously kept in view" in this edition, is the effort to "produce a work which would occupy a position somewhere between a technical dictionary of medicine and one intended merely for the domestic treatment of the commoner ailments." It is further "hoped that these articles will be found useful to district nurses, ship captains, dwellers in remote districts and others who may be called upon to treat the suffering in the absence of trained supervision."

When the book is judged in the light of what it purports to be, it is found to be an excellent compilation of well-known medical facts and descriptions of well-worn medical terms.

Judged in the abstract as a medical dictionary, it falls far short of perfection through its incompleteness.

By its purposeful omission of many of the less common medical words and many of those recently coined, it will as often disappoint as reward a medical man in its use.

In a general way the book seems to fall into that class of medical books whose chief attraction lies in short cuts to medical knowledge, of which the English seem so fond. A. A. H.

MEDICAL JURISPRUDENCE. A STATEMENT OF THE LAW OF FORENSIC MEDICINE. By ELMER D. BROTHERS, B.S., LL.B., member of the Chicago Bar; Lecturer on Jurisprudence in the Medical and Dental Departments of the University of Illinois and in John Marshall Law School. Pp. 299. St. Louis: C. V. Mosby Co., 1914.

THE contents of this book is the fruit of twenty years of study, practice, and teaching on matters of a medico-legal nature. Its writing was incited by the lack of a satisfactory book of reference for medical students in their study of the legal aspects of medical practice.

The author has devised a ready and interesting means for both the prospective and practising medical man's learning, just what his legal medical responsibilities are. To one who has not given the subject thought, it comes as a rather startling fact that as physicians we are unconsciously entering into numbers of definite contractural relations daily which, upon default on our part, have their legal redress. On the other hand it is comforting to be told just what comprises a complete fulfilment of these obligations and responsibilities. A knowledge of the patient's contractural status and his obligation is also valuable, and will give the physician assurance in standing on his rights.

In addition to this general information of every-day use to

internist, surgeon, and specialist alike, medical matters with more special legal bearings are discussed. These include expert testimony, privileged communications, civil and criminal malpractice, insanity, wounds, blood stains, crimes, etc.

It may be that lessons learned from this book will forestall a future learning from the book of bitter experience. One sees, however, that the law has not been one-sided: it insists equally on the rights as on the responsibilities of the physician, and it behooves every medical man to know just what his rights are.

A. A. H.

SURGERY OF THE BLOODVESSELS. By J. SHELTON HORSLEY, M.D., F.A.C.S., Surgeon-in-Charge of St. Elizabeth's Hospital, Richmond, Va., etc. Pp. 304; 89 illustrations. St. Louis: C. V. Mosby Company, 1915.

THE preface of this volume says: "It has been the aim of the author to present the scientific and the laboratory features of vascular surgery and particularly its practical aspects that may be of interest both to the surgeon and to the general practitioner. Therefore the treatment of hemorrhage, pathological and traumatic, and such subjects as aneurisms, thrombosis and embolism, congenital nevi, varicose veins, and hemorrhoids are described as well as the history and technique of suturing bloodvessels and transfusion of blood." An aim such as this requires the use of cannister or of shrapnel at close range to hit all the individuals for whom the discharge is intended. This is the weak point of the book. The only subjects which are described better than in other works relate to mere matters of technique, especially the suture and anastomosis of bloodvessels and the transfusion of blood. These chapters are excellent, the illustrations above the average, and the author speaks with authority born of ample experience, yet with pleasing modesty. It remains to be seen, whether the recent introduction of the method of transfusion by the aid of citrate solutions will entirely replace those here described. Most of the other subjects are treated quite superficially, both from the scientific and laboratory stand-point and from the purely technical side. The chapters on hemorrhage are particularly lacking in both respects. An example is found in the totally inadequate, indeed, we may say the dangerously inadequate, account given of Momburg's method of hemostasis. The best chapters, apart from those already mentioned, are those on thrombosis and embolism. Yet here again otherwise excellent work is marred by an attempt at all inclusiveness. Surely, it was not necessary in a work purporting to deal with the surgery of bloodvessels to include illustrations and elaborate descriptions of intestinal resection and anastomosis, although the ostensible

excuse for such inclusion is found in the fact that these procedures may become requisite in cases of mesenteric thrombosis or embolism. Why not have described amputations which may be required in various lesions of the limbs which are included? A. P. C. A.

THE DIFFICULTIES AND EMERGENCIES OF OBSTETRIC PRACTICE. By COMYNS BERKELEY, M.A., M.D., M.C. (Cantab.), F.R.C.P. (Lond.), M.R.C.S. (Eng.), Obstetric and Gynecological Surgeon to the Middlesex Hospital; Surgeon, Chelsea Hospital for Women, Senior Obstetric Surgeon, City of London Lying-in Hospital, etc., and VICTOR BONNEY, M.S., M.D., B.Sc. (Lond.), F.R.C.S. (Eng.), M.R.C.P. (Lond.), Assistant Obstetric and Gynecological Surgeon to the Middlesex Hospital, Surgeon, Chelsea Hospital for Women, etc. Second edition. Pp. 807; 302 illustrations. Philadelphia: P. Blakiston's Son & Co., 1915.

THE authors have given in an almost encyclopedic form a description of and rules to govern the treatment of difficult and complicated cases in obstetrics. The affections of the various organs and body systems are taken up in order, and are followed by chapters on the diseases of the ovum, on hemorrhage, and on the various forms of dystocia and the abnormal puerperium. The well-written chapters on obstetric surgery embraces practically all operations performed during the process of reproduction. The diseases of the newborn child and the artificial feeding of infants are discussed in the concluding chapters.

That the scope of the book may be better recognized, it is noted that under diseases of the intestinal tract fourteen topics are discussed; under disorders of the nervous system there are grouped nineteen subheadings; the diseases and injuries of the newborn child include references to sixty-one conditions; we find a reference to ankylostomiasis, another to the marriage of first cousins. However, to those rare complications as hepatic abscess and rupture of splenic aneurysm during pregnancy but little space is given; we also find a full and reliable discussion of tuberculosis and syphilis. For obvious reasons no mention is made of the physiology and management of normal cases. Difficult labor is dealt with fully, all forms of fetal and maternal dystocia, and the methods for their relief being taken up at length. The authors mention six conditions where craniotomy in the living child is indicated. Probably only a few men possess fingers of the length of those on the hand illustrated in Fig. 156. The position of the hand illustrated in Fig. 169, portraying the operation of internal podalic version, is directly opposite that recommended in the text. The corkscrew position

of a pregnant woman for routine examination, Fig. 1, must necessitate some considerable ability of orientation on the part of the examiner. Chloroform, favored by the authors in labor with cardiac disease, is not the anesthetic of unanimous choice among American obstetricians, who also do not wholly agree with the authors as to the value of routine vaginal douching after normal labors.

Apart from these criticisms the book will be found to be fairly descriptive, and certain to be a practical and valuable aid to every practitioner of obstetrics. P. F. W.

THE NEWER PHYSIOLOGY IN SURGICAL AND GENERAL PRACTICE. By A. RENDLE SHORT, M.D., B.S., B.Sc. (Lond.), F.R.C.S., Hunterian Professor, Royal College of Surgeons, etc. Third edition; pp. 256. New York: William Wood & Co., 1915.

THIS book is an interesting collection of the most recent work bearing upon problems in physiology of direct practical importance to the physician and surgeon. The light of modern research is utilized to explain many of the phenomena met with in common practice. Among the most important questions discussed are the relation of vitamines to the occurrence of nutritional disturbances, such as beri-beri, scurvy, and rickets; surgical shock; the growth of bone; ductless glands; clinical physiology of the alimentary canal; the hemorrhagic diathesis; acidosis and diabetes; nerve regeneration.

In many of the conditions discussed, fallacies are exposed by the ray of science and valuable suggestions for rational treatment given. Thus the book is not only of interest in the abstract, but should be a direct practical aid to the practitioner in his daily work. R. H. I.

PAINLESS CHILDBIRTH. By MARGUERITE TRACY and MARY BOYD. Pp. 316; 19 illustrations. New York: F. A. Stokes Company, 1915.

THE recent sensational exploitation of twilight sleep by means of department-store lectures and feature articles in the popular monthly magazines has been followed by its presentation in book form. The title suggests the Utopian dream of the gravid woman. In a more or less haphazard manner the authors have combined references to foreign and American medical literature, remarks and abstracts of discussions on the topic and narratives of personal experiences with the method by several women. Much space is given to a description of the maternity clinic at Freiburg, and

one wanders through many pages in following the development of the Dammerschlaf. The last third of the book consists of translations of articles by Kroenig and Gauss on this method of analgesia-amnesia in parturition. The illustrations serve no purpose, portraying, for the most part, a healthy looking set of infants and children. The book can hardly be recommenced as a scientific, impartial, or convincing presentation of the subject. P. F. W.

TEXT-BOOK OF LOCAL ANESTHESIA. By D. GEORGE HIRSCHEL, Assistant in Heidelberg Clinic. Pp. 181; 103 illustrations. London: William Wood & Co.

THE author has endeavored to write a comprehensive treatise on the subject of local anesthesia. He has, according to his own statement, attempted to sift out the chaff and retain the wheat. This he has accomplished, in the reviewer's opinion, too well. He treats fully and well the anatomical features, and the work is replete with many excellent anatomical and applied surgical cuts, with a special reference to the nervous anatomy and the regions supplied by each group.

The technique of administration, the actual area to be anesthetized, and the extent of the uses of the method are all fully laid down.

All of us know that novocain plus some adrenal extract is the best drug on the market for local anesthesia, but there are also other very useful drugs, which the author either mentions once or not at all, a mistake of his to term his work one on "local anesthesia" and not, "novocain" anesthesia. E. L. E.

CAUSES AND CURES OF CRIME. By THOMAS SPEED MOSBY, Member of the American Bar. Pp. 353; 26 illustrations. St. Louis: C. V. Mosby Co.

THIS is a very interesting book. The author attempts to treat in a popular style the causes and cures of crime. Naturally such a large subject can only be discussed in a sketchy manner in such a short book as this, yet it is so well done, that the reader gains an excellent idea of the subject.

The author throughout takes the point of view that the whole question is medical and not legal. In giving the various causes for crime, he first takes up the question of seasons and develops the interesting fact that hot weather seems to have an effect chiefly

in increasing crimes of impulse, while in cooler climates crimes against property are most frequent. In discussing the social factors of crime he takes up the law's delay and the very important question of law-making, pointing out the fact that with each new law new crimes are created. City life is much more prone to lead to crime than country life. He then takes up the influence of newspaper publicity upon the causation of crime, citing the well-known Thaw case. It is an interesting fact that crime is less frequent in poverty stricken nations than in those that are prosperous. He takes issue with those who do not believe that religion has its influence on the cessation of crime and especially criticises the views of Lombroso. He points out that the "refinements of life corrupt while they polish the intercourse of the sexes. The gross appetite of love becomes more dangerous when it is elevated, or rather, indeed, disguised, by sentimental passion." A point of view which cannot altogether be agreed with. From this it can be readily seen that the book is interesting and well worth reading.

T. H. W.

RECREATIONS OF A PHYSICIAN. By A. STUART M. CHISHOLM, G.P. Pp. 328. New York: Putnam's Sons.

IN these days of bustle and hustle, recreation is imperative for the physician, though much be sacrificed in the obtaining thereof. The title of the book under review seemed to make a strong appeal, and it was immediately conjectured that here would be found practical suggestions for the busy doctor, suggestions as to play, and hence recreation, from one who had had not only the time to think and to write about the subject, but also to experience the recreations which he was to offer to us. One needs to read but a few pages to realize how far wrong this conjecture is, for the title, *Recreations of a Physician*, indicates a volume of collected essays, read and printed by Chisholm, presumably as a part of his recreation scheme. Therefore, far from receiving any helpful hints as to how to recreate, one is baffled by the thought that since these splendid essays are merely recreation pastimes, what must and should be the product of a mind at work?

The essays are ten in number, and are uniformly good, dealing generally with subjects of historical interest, as might be expected, when the author's name is recalled. The "Banquo" contains some delightful views on the various readings of Shakespeare's text, the explanation of corrupted passages by different commentators, and the emendations that have been proposed to clarify obscure portions of the text; but why it is called "Banquo" is incomprehensible, even after reading "The Symbolism of Names," the next essay. In this essay occurs a delightful tale by Quevedo of Pontius Pilate,

which has the charm of being "new," not chronologically, but in the sense of narration.

To the reviewer's mind "The Inherent Spirit of Medicine" is the best essay. In it is a defence of the profession of medicine with an exposition of its ideals, which every practioner, but especially every medical student, should read. "The study of medicine is an entrancing subject; its practice requires an array of virtues whose mere contemplation staggers the mind. One must meet violence with gentleness, ingratitude with equanimity, insult with fortitude, slander with silence. The physician's life is a daily exemplification of the Goldern Rule. The very sensitiveness that inspires sympathy with pain and misery is a weapon in the hands of ignorance and malice wherewith they deal dreadful wounds which must be endured silently. Resentment can have no place in the physician's mind. Equanimity must be maintained in the face of misapprehension and abuse." The medical student needs no better vade mecum than this.

To those seeking recreation, to those desiring information, and to those eager for instruction, this volume of Chisholm is warmly recommended. E. H. G.

HAND-BOOK OF PHARMACOLOGY. By CHARLES WILSON GREENE, A.B., A.M., PH.D., Professor of Physiology and Pharmacology, University of Missouri; Member of American Association of Anatomists, American Physiological Society, etc. Pp. 396; 70 illustrations. New York: William Wood & Company, 1914.

THIS hand-book is a presentation of the subject of pharmacology in the restricted sense of that term. In it Greene does not discuss drugs in their relation to diseased conditions but only the reactions of the normal body to drugs and drug agents. In other words, he distinctly separates pharmacology from therapeutics just as physiology is separated from pathology. This, he claims, will enable the student, for whom the book is primarily written, to get the principles of the subject of pharmacology without being burdened and confused by a mass of matter on practical materia medica and therapeutics.

He classifies his presentation of the subject according to the physiological reactions of particular groups, devoting a single chapter to each group or series of drugs, and in each case detailing the action of only the characteristic one or ones. Except for a brief introductory paragraph on the history and chemistry of the drug in each case the entire chapter is devoted to the reactions of the normal body to the drug, these effects first being outlined in italics, then presented in detail, and finally concisely summarized. This arrangement has pedagogical advantages and makes the book convenient for ready reference. T. G. M.

COXA VARA. By R. C. EMSLIE, M.S., F.R.C.S., Orthopedic Surgeon to St. Bartholomew's Hospital, London. Pp. 35; 34 illustrations. Oxford University Press.

THIS monograph of about ten thousand words is devoted to a scientific investigation of coxa vara. The facts here presented are based upon a clinical study of cases, anatomical specimens, and radiographic investigations. There is a brief discussion of the development of the neck of the femur and anatomical coxa vara. Adolescent coxa vara is clearly outlined, with a brief summary of the etiological factors, symptoms, and radiographic findings. Infantile coxa vara is briefly discussed and moderate emphasis placed upon the causative factors. The pathological findings, radiographic and histological, are the result of the author's personal work. The treatment of this condition as outlined by the author is comparatively brief, stress being laid chiefly upon correction by apparatus and operative procedures. Although this work is comparatively brief it has a decided scientific value. W. J. M.

ALVEOLODENTAL PYORRHEA. By CHARLES C. BASS, M.D., Professor of Experimental Medicine at the Tulane Medical College, and FOSTER M. JOHNS, M.D., Instructor in the Laboratories of Chemical Medicine at the Tulane Medical College. Pp. 167; 41 illustrations. Philadelphia and London: W. B. Saunders Company, 1915.

THE recent discovery of the cause of pyorrhea alveolaris and the intense interest that it has aroused makes particularly appropriate the appearance of a monograph upon the subject by the two authors who, independent of the investigations of Smith and Barrett, published, shortly after these authors, the result of their work upon the same line, showing that the Endameba bucallis is pathogenic. These two groups of investigators independently came to the conclusion that the organisms are pathogenic because of the favorable effects upon the lesions of the disease produced by the action of emetin. These results have subsequently been corroborated by numerous observers so that at the present time an annoying, disagreeable and sometimes serious disease has been deprived of much of its former power of harm.

The monograph is divided into nine chapters, of which those dealing with the etiology, morbid process, symptomatology, diagnosis, treatment, and prophylaxis are the most important. Probably the most interesting chapter is the one upon prophylaxis in which rather radical views are expressed as to the necessity of the tooth brush and of tooth powders.

The book can be recommended as an admirably complete and accurate summary of our knowledge of the subject. Its chief fault, perhaps, is the too obvious effort to spread out as much as possible the subject matter, to make the number of words as few to the page as possible, to make the print much larger than necessary and in fact to give the impression that a great deal more has been said upon the subject than is actually so. J. H. M., Jr.

Les Techniques Anatomo-pathologiques du Systeme Nerveux. Anatome Macroscopique et Histologique, par Gustave Roussy, Professor Agrege, Chef des travaux d'anatome pathologique a la Faculte de Paris, et Jean Lhermitte, Ancien Chef de laboratoire a la Faculte de Paris. Pp. 55; with illustrations. Paris: Masson et Cie., 1914.

This is a small volume devoted to the subject of neurological technique in the laboratory. The authors first treat of the method of hardening the brain, and describe Marie's method of cutting it. The methods of fixing, embedding, and staining the tissues are described in detail. Separate chapters are given to the subject of staining each element of the nervous system, the cell, axis-cylinder, neuroglia, etc. The authors are thoroughly conversant with the newest methods of staining. The arrangement of the book is excellent. A summary of the procedure is found at the end of each staining method. The book is a good practical manual to have at one's elbow in the laboratory. S. L.

Anatomy and Physiology for Nurses. By Amy E. Pope, Co-author of "Practical Nursing" etc. Pp. 554; 135 illustrations. New York: G. P. Putnams Sons.

At present, when the nurse plays so prominent a part in Social Service, it is important that she should understand the reasons for the orders that she gives and transmits to the patients in her care. For this purpose, the present volume, by reason of its completeness, is admirably adapted. It is attractively gotten up and would make an excellent text-book for any nurses' training school. S. J. R.

PROGRESS

OF

MEDICAL SCIENCE

MEDICINE

UNDER THE CHARGE OF

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Detoxication of Diphtheria and Tetanus Toxins.—J. SCHUMACHER (*Deutsch. med. Wchnschr.*, 1915, xli, 310) reports the results of experiments he has performed to protect animals from fatal doses of diphtheria and tetanus toxins. Sieber has shown that abrin, diphtheria, and tetanus toxins can be rendered non-toxic by previous treatment with hydrogen peroxide. The latter substance is not suitable for intravenous use. Schumacher selected ammonium persulphate, which is also a strong oxidizer, for his experiments. It is relatively non-toxic. The experiments were performed upon guinea-pigs. The animals were given eight times the lethal dose. The controls all died within twenty-four hours after receiving the toxins. In a second series of animals, 2 c.c. of 5 per cent. ammonium persulphate was allowed to act upon the diphtheria toxin in the syringe for ten minutes before the injection was made. These animals showed no symptoms whatever. Intravenous administration of 2 c.c. of 5 per cent. ammonium persulphate solution ten minutes before giving the toxin delayed the death of the animal twelve hours. Similar results were obtained with tetanus toxin. A more detailed study of this question will appear in a later communication.

Leukopenia and Aneosinophilia following Typhoid Vaccination.—FRIEDA SCHNEIDER (*Deutsch. med. Wchnschr.*, 1915, xli, 393) confirms the findings of others to the effect that agglutinins for typhoid bacilli appear in the blood following antityphoid vaccination. She has found agglutination positive in dilutions of 1–160 to 1–320. This finding, of course, renders the Widal test of little value in suspected typhoid in those who have been vaccinated. The author investigated

other blood changes found in typhoid fever, *i. e.*, leukopenia and aneosinophilia. Examination showed that both of these phenomena made their appearance within a few days after the third injection of typhoid vaccine and that they may persist for several months. This should be borne in mind in the investigation of vaccinated individuals.

Agglutination of the Spirocheta Pallida.—A. KISSMEYER (*Deutsch. med. Wchnschr.*, 1915, xli, 306) has investigated the possibility of an agglutinin formation in syphilitics, since the literature of the subject is rather contradictory. In studying human syphilitic sera, the author was able to demonstrate an agglutination in each stage of the disease, though the phenomenon was an inconstant one. He looks upon agglutination in dilution of 1 to 100 occurring after two to three hours in the incubator at 37° as positive. Occasionally, a non-syphilitic serum will cause agglutination in dilutions of 1 to 50. Most positive sera will also cause agglutination in dilutions of 1 to 100. In one patient in the primary stage of the disease, agglutinins appeared in the blood before the Wassermann reaction became positive. In congenital syphilis the agglutinin reaction was strongly positive in patients under twelve years of age. With increasing age there was a decrease in the intensity of the reaction. By injection of killed spirochetes into rabbits the author was able to produce sera having very strong agglutinating power. The effect of treatment on the presence or absence of agglutinins in the sera of syphilitics is under investigation.

The Bacterial Therapy of Typhoid Fever.—Encouraged by the reports of Ichikawa and of Kraus and Mazza, M. RHEIN (*München. med. Wchnschr.*, 1915, lxii, 427) has tried the bacterial therapy on a series of thirty-three patients suffering with typhoid fever. He prepared his vaccine as follows: To 5 c.c. of Halle typhoid vaccine and 0.5 gm. pure phenol, physiological salt solution was added to a volume of 100 c.c. Of this preparation he employed 0.6 to 1.4 c.c. diluted with 8 c.c. physiological salt solution for intravenous injection. Within one to two and a half hours a rise in temperature and a chill of varying severity and duration (five to thirty minutes) occurred in 94 per cent. of the cases. Injections were made only in patients in whom the diagnosis was clear clinically or bacteriologically. Three of the patients treated showed paratyphoid bacilli A in the blood. Patients exhibiting the step-like curve of defervescence were excluded, as were also those suffering with complications such as pneumonia, pleurisy, nephritis, or myocarditis. Status typhosus, with delirium and bronchitis, but with good pulse, was not looked upon as contra-indication for injection of vaccine. Thirty-three patients received the injections and six of these had a second injection. In 9 cases there was a critical fall of temperature within one day after the first injection and in 1 case after the second. In 13 cases a favorable effect on the temperature curve was noted after the first injection and in 3 cases after the second injection. In 8 patients there was no effect whatever on the temperature curve, and in 2 of these a second injection was also without visible effect. On the other hand, 3 patients who were unaffected by the first injection reacted favorably after a second. In the 3 cases of paratyphoid fever a critical fall of temperature was not observed, but

there was a gradual lowering in the fever. Of the patients injected, 3 died; in none could the death be attributed to the injection. Of all the patients treated, only 1 suffered from intestinal hemorrhage, which occurred six days after the injection. Other complications were not observed in the injected patients. No evidence of collapse even in patients with weak pulse was seen. The reaction of the patient to the injection was studied with relation to the following clinical signs or symptoms: Bronchitis, palpable spleen, roseola, Diazo reaction, leukocyte count, eosinophile count, and bacteriemia. A favorable response to the injection could not be correlated with any of these. However, after crisis, comparatively many eosinophiles (100 to 200 per c.mm.) appeared in the blood in twenty-four hours in spite of the leukopenia, and the Diazo test remained positive as long as three days after the temperature was normal. It was noticeable that with larger dosage (4 c.c.) the crisis was of shorter duration. Since no ill effects followed the injection, and, on the contrary, in 48 per cent. of the cases the fever was shortened and in 30 per cent. cure followed within two days, the author feels that in every case of uncomplicated typhoid fever, bacteriotherapy should be tried.

A Test for Antithrombin in the Blood.—There have been thus far but few estimations of the antithrombin content of the blood. Most of them have been made by Howell, who has used a method which he has devised, and though it is satisfactory, it is not simple and not suited to wide clinical use. The substances required in the test demand considerable experience in their preparation. Hess (*Jour. Exper. Med.*, 1915, xxi, 338), therefore, has devised a test, the chief advantage of which is its simplicity and the fact that it does not require a difficult preparation and maintenance in a pure state of fibrinogen and thrombin. The principle involved consists essentially in titrating the antithrombin against normal human plasma. The method briefly is as follows: 9 c.c. of blood are put into 1 c.c. of 1 per cent. sodium oxylate. The mixture is centrifuged and the plasma removed in the usual way. The plasma is then recalcified by adding 2, 3, 4, and 5 drops respectively to a 0.5 per cent. calcium chloride. By this method one ascertains the general coagulability of the plasma and the optimum amount of calcium for this particular plasma. It is well known that heating plasma to 60° C. destroys prothrombin and coagulates fibrinogen. If now this plasma is filtered the filtrate contains antithrombin, the strength of which can be ascertained as follows: some normal plasma is prepared in exactly the same way as is the oxylated plasma to be tested, 5 drops of which are put into five carefully cleaned vials. The first tube serves as a control. To the second and third tubes are added three and five drops respectively of normal antithrombin; to the fourth and fifth tubes equal amounts of antithrombin that is to be tested. The volume of all the tubes is made equal with normal salt solution. The tubes are allowed to stand fifteen minutes. The plasma is then recalcified by the addition of 0.5 per cent. calcium chloride. Antithrombin is judged to be in excess when a marked delay in coagulation is brought about in the tubes to which it has been added as compared to the coagulation in the control tube.

Report on the Allen Treatment of Diabetes.—HILL and SHERRICK (*Boston Med. and Surg. Jour.*, 1915, clxxii, 696) report in the present article their results in a small series of cases of diabetes which have been treated according to the method recommended by Allen. In brief, their routine is to put the patient first on a house diet for two days, thus determining his tolerance for ordinary diet and the severity of his diabetes. On the third day the patient is put to bed and given nothing but black coffee with one ounce of whisky every two hours from 7 A.M. to 7 P.M. This diet represents an intake of about 800 calories. Provided there is much acidosis, as indicated by the amount of diacetic acid and acetone in the urine, sodium bicarbonate is given. Patients are kept on this regime until sugar-free. This usually requires from two to three days, during which time there is usually a slight loss of weight and relief from such symptoms as itching and excessive thirst. They have never observed any indication of acid poisoning in cases treated by this method. As soon as the patient is sugar-free he is given a single "vegetable day," after which the diet is slowly raised, increasing first the fat, then the protein, and lastly the carbohydrate. Fat is never raised above 200 grams and the caloric intake rarely above 2200. They emphasize three important things in the treatment: (1) not to worry if the patient loses weight; (2) not to raise the diet too quickly after starvation; (3) pay just as much attention to the protein intake as to the carbohydrate. Included with the article there is a series of diets which have been carefully figured out, particularly to meet the requirements of patients after they have left the hospital. These will no doubt prove valuable.

Uric Acid Excretion in a Case of Total Occlusion of the Pancreatic Duct.—When a patient in the medical service of the Johns Hopkins Hospital was found to have a complete absence of pancreatic secretion in the intestinal tract the idea was conceived by D. W. ATCHLEY (*Arch. Int. Med.*, 1915, xv, 655) that by feeding thymus gland and estimating the increased output of uric acid in the urine the possible necessity of the pancreas to any stage of nuclear digestion could be conclusively demonstrated. The patient, therefore, was put on a purin-free diet, and for four days the uric acid was determined by means of the Folin-Schaeffer method. On the fifth and sixth days the patient was given in addition 150 grams of fresh calf's thymus with the noon meal. The results showed there was practically a quantitative recovery of exogenous uric acid, a finding which conclusively demonstrates that there may be digestion of the thymus nuclei, with the production of uric acid in the urine, in the absence of both bile and pancreatic juice. This finding as well as similar ones in other cases, definitely points out the worthlessness of the Schmidt-nuclear test for pancreatic function.

Renal Irritation in Relapsing Fever.—L. JARNO (*Wien. klin. Wchnschr.*, 1915, xxvii, 416) has studied the urine in a series of 170 uncomplicated cases of relapsing fever. Daily examinations were made and strikingly constant changes were found. The first day of the disease only a trace of albumin may be found in the urine. On the second day of the attack there is 0.5 to 1.5 gms. of albumin per liter, and at the same time

numerous granular casts appear. During the first attack of fever these changes remain quite constant. On the third afebrile day, more rarely on the second, there is a gradual decrease in the number of granular casts and in the amount of albumin. By the fourth day the urine is again normal. The urinary findings in the second febrile attack are in every way similar to those in the first, and the urine also clears up in the afebrile period in the same way. If a third and fourth attack occur the albuminuria usually returns, but the granular casts are generally much less abundant than in the first two attacks. The albuminuria, however, disappears early. It seems evident that there is a severe renal injury at the beginning of the disease, but as the infection continues, the kidneys seem to become more resistant to the harmful agent. Exceptional cases were noted where the urinary changes were in some instances less pronounced, in others more severe. In one instance, chronic nephritis remained.

Vaccine Therapy in Typhoid Fever.—In answer to inquiries from Prof. Paltauf, BIEDL and H. EGGRETH (*Wien. klin. Wchnschr.*, 1915, xxvii., 125) have given their experiences with the vaccine treatment of typhoid fever. Paltauf publishes their letters in full. Biedl has treated a series of 22 cases of severe typhoid fever in the beginning of the second week; the blood culture was positive in all of these. Of this number 2 are excluded. Both were men with high fever, status typhosus, and recurring epistaxis, the last nose-bleed occurring one day before injection in each case. Two hours after injection of the vaccine there occurred in each patient an uncontrollable hemorrhage from the nose which led to death. Of the remaining 20 cases, 11 received Vincent's vaccine (typhoid bacilli killed with ether), first 100,000,000, later 250,000,000 to 300,000,000 in 2 c.c. of salt solution, and 9 were treated with Besredka's vaccine intravenously (sensitized living typhoid bacilli) in doses of 250,000,000 to 300,000,000 in 2 c.c. of salt solution. The end result was as follows: Of the cases treated with Vincent's vaccine there were 3 who died; 2 received subcutaneous injection and died fourteen and eighteen days respectively after the injection; 1 treated intravenously, after an initial favorable response, died in the third week from severe bronchopneumonia and heart weakness. Evidently none of these deaths were due to the vaccine. The remaining 8 patients recovered. All of the patients treated with Besredka's vaccine recovered. Biedl's impressions were decidedly favorable. Following the intravenous injection there was a rise in temperature in one to two or three hours from 39° C. to 40° or 41° C., in one case even to 42° C., followed in twelve to eighteen hours by a critical fall of temperature to normal. No signs of collapse were observed; indeed, in two cases the injection was made when the patients were in a state of collapse with a pulse which could not be counted. Within three hours the temperature rose, the pulse became much fuller and 100 per minute. Following crisis the patients felt much better. The patients treated with Besredka's vaccine remained afebrile. Some of the other patients had slight evening elevations of temperature; in none above 38° C. Eggreth treated a series of 43 cases of typhoid fever with a single intravenous injection of 0.5 to 1.0 c.c. of Besredka's vaccine. Subcutaneous injections had produced no result. In 34

cases of Eggreth's series a critical fall of temperature followed within three to twelve hours after the injection, frequently with profuse sweating and marked relief of headache and delirium. Of these patients 31 remained afebrile; the remaining 3 developed fever after twelve, fourteen, and fifteen days, due respectively to endocarditis, to bilateral purulent parotitis, and to suppurative osteoperiostitis. The majority of the patients received the injection between the seventh and sixteenth day of the disease. In a group of 8 cases in the fourth and fifth week of the disease, suffering with broncho- or pleuro-pneumonia, the injection was ineffective. The forty-third case died three hours after the injection. An autopsy was performed which revealed typical typhoid lesions in the small intestine, pneumonia, and myocarditis. The patient had been brought to the hospital unconscious and desperately ill, and a poor risk. Paltauf utters a word of warning, as he has heard of a few instances of collapse following the intravenous use of Besredka's vaccine.

The Bacteriology of Appendicitis.—Rosenow (*Jour. Infect. Dis.*, 1915, xvi, 240) reports the results of his observations and experiments, which seem to indicate that appendicitis, in the absence of foreign bodies, is very frequently of a hemtogenous origin and secondary to some distant focus of infection. The organisms most commonly found in the distant foci are streptococci, and the disease develops when, for some reason or other, the organisms have acquired an affinity for the appendix and at the same time gain entrance into the circulation. These findings further bear out the author's theory that a focus of infection is to be regarded not merely as the place of entrance of bacteria, but also as the place where they may acquire the power necessary to infect distant organs and tissues. The importance is emphasized, therefore, of a thorough search for and removal of possible foci of infection, from which appendicitis may originate, and that this may occur in clinical experience is indicated by the not infrequent occurrence of the disease, at times almost in an epidemic form, when throat infections are particularly prevalent.

The Factors of Coagulation in Pernicious Anemia.—Drinker and Hurwitz (*Arch. Int. Med.*, 1915, xv, 733) have studied the various factors in a series of cases, including seven of pernicious anemia. They emphasize again the essential characteristics of this disease and especially those cases which are distinguished chiefly by failure to regenerate blood cells and by a tendency to bleed. In a typical case reported in full, they were able to note that the coagulation time was distinctly prolonged once only; that the bleeding time was always long, and that the platelets were always far below normal, nor were they consistently increased by transfusion. Antithrombin was consistently normal, as was fibrinogen, and neither was affected by transfusion, though this procedure tended to cause a slight transient rise in pro-thrombin, which was otherwise constantly decreased. This diminution in prothrombin is not great and is probably unimportant, provided active regeneration is in progress. The other normal findings occurred even in the presence of extremely low cell counts.

Nitrogen Metabolism in Dermatoses.—TIDY (*Quart. Jour. Med.*, 1915, viii, 129) gives a general review of the earlier studies and opinions upon this subject, as well as the results of more recent researches, which have been carried out particularly in cases of erythema, urticaria, and cases showing scaling or bullous eruptions. As a result of the investigations, it seems apparent that the changes in the nitrogenous excretion in various skin diseases are the result of the condition of the skin and are not connected with the cause of the disease. The retention of nitrogen is due to an abnormal nitrogen excretion by the skin, and changes in nitrogen excretion may precede the eruption as well as survive it. In view of these findings it is distinctly reasonable and advisable that a low protein diet be tried in these forms of dermatitis which are known to be associated with disturbances of the nitrogen excretion.

SURGERY

UNDER THE CHARGE OF

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The Treatment of Gun-Shot Aneurysms in Special Sites by Artificial Strengthening of its Walls.—HABERLAND (*Zentralbl. f. Chir*, 1915, xli, 281) reports a case of traumatic aneurysm due to a gun-shot wound at the junction of the external iliac and femoral artery, which increased in size slowly and was operated on ten weeks after the injury. The method employed applies only to peripheral aneurysms of moderate size. There must be many cases in which such an operation will preserve the continuity of the lumen of the injured artery, and prevent further growth and rupture of the aneurysm, as well as relieve the pain. In this case the aneurysm was 8.5 by 5 cm. in size, pulsated strongly and its upper border could not be clearly outlined by palpation over Poupart's ligament. A large skin flap was made with its base above Poupart's ligament and the aneurysm about in the middle of the exposed area. Care was necessary in the separation of the flap, as only thin scar tissue separated the wall of the aneurysm from the skin in the region of the gun-shot wound. A flap was made from the fascia external to the aneurysm with its base along the border of the aneurysm, large enough to permit it to be turned inward and easily cover the aneurysm. It was then fixed in position by close silk sutures and the skin flap sutured in position over it. Healing was good. Three months later no further growth of the aneurysm could be detected.

Disinfection of the Hands and Abdominal Skin Before Operation.—McDonald (*Surg., Gynec., and Obst.*, 1915, xxi, 82) says that his experiments show the germicidal strength of alcohol to be of no great value. Its virtue in cleansing the hands is exerted more as a chemical solvent than as a germicide. Though a solvent of free fatty acid, it will not dissolve the fat as found in the skin, *i. e.*, the glyceride. The ideal skin disinfectant should be a fat solvent. Alcoholic solutions of iodine give only superficial disinfection, while with solutions of iodine in fat solvents such as tetrachloride or chloretone, there is complete penetration, as has been shown by McDonald's carbon tetrachloride 2 per cent. iodine solution. During a period of ten years, McDonald has tried many hundreds of solutions and germicides and has compared one with another as to results. His final solution has the following parts: Acetone (commercial), 40 parts; denatured alcohol, 60 parts; and pyxol, 2 parts, the latter being the germicide. The method of disinfection advised is simply to treat the hand in this solution in a vessel sufficiently large to admit the hands and rub, with a nail brush for the nails and a gauze cloth for the arms, to aid the solution in permeating every crevice. The immersion should last one minute. This solution has the following advantages: It is reasonably cheap; does not irritate the skin; contains a fat solvent, acetone, causing the solution to penetrate; the alcohol is a good vehicle; the solution contains a strong germicide which is twenty times as germicidal as carbolic acid in equal strengths and this germicidal value is not impaired by the alcohol or acetone. It has the germicidal value of phenol in 40 per cent. solution (if such a solution could be made), plus the germicidal action of the solvent. McDonald has used it for more than a year with uniformly satisfactory results. In the sterilization of the skin of the abdomen, it is rubbed on for two minutes before operation after the patient is under ether without any preliminary washing. Since it is possible to sterilize the hands so thoroughly and in such a quick and easy fashion, there does not seem to be any further advantage in using rubber gloves, for the hands can be sterilized after contamination in less time than it takes to remove the contaminating gloves and put on a clean pair. Since he has not used gloves, McDonald has, however, fortified his skin by means of a hand varnish which he has described in another journal.

Report on Skin Disinfection by McDonald's Solution.—McMullin (*Surg., Gynec., and Obst.*, 1915, xxi, 87) had the field of operation prepared, if possible, on the evening before the operation, by shaving and washing with green soap, water and alcohol. It was covered with a dry sterile towel and left until the next day. At operation the site was treated by rubbing with McDonald's solution for about two minutes. The hands of the assistants and operator were prepared by scrubbing with soap water and alcohol, followed by two minutes' treatment with McDonald's solution. He has operated, with this preparation, on over 276 major cases, practically all laparotomies and hernias. In emergency cases the soap and water scrubbing of the operative field was dispensed with and only McDonald's solution used. In these cases there were 7 skin infections. Excluding those cases in which infection

was obviously due to some extraneous causes, there remained but 3 cases in which there was any skin infection, which might be attributed to improper disinfection of the skin. From this analysis of his experience, McMullin believes this solution to be the most efficient for skin disinfection at our disposal at the present time. Healing under its use seems to take place more readily than under the iodine skin disinfection. McMullin believes that wound repair is accelerated by at least three days, when compared to that following the iodine skin disinfection. Applied to the skin of the hand, the solution is unirritating and is almost ideal as a skin disinfectant.

Sterilization of the Skin by McDonald's Solution.—STANTON (*Surg., Gynec., and Obst.*, 1915, xxi, 89) says that water, up to 10 per cent. or more, does not interfere with the action of the solution. Hence it is possible to use it on wet skin surfaces, such as those recently lathered for shaving, or the operator's hands after he has scrubbed them with soap and water. The solution is itself an excellent cleansing medium and so harmless that it can be used actually to scrub the patient, whereas tincture of iodine should not be rubbed in. It can be used before the patient is anesthetized without danger of complaint, as it is not irritating. It does not stain skin surfaces and dries very quickly, so that it is not necessary to protect peritoneal surfaces from contact with the skin, as in the case of iodine skin treatment. Stanton had only 2 infections in 240 otherwise clean cases, a record never before equalled in his work. During the year 1914 there was not one drop of pus collected in a clean case operated upon by him. Such results are significant and at least show that the method of skin preparation was efficient. Even more important was the fact that the wound healing was without evidence of chemical irritation, without peeling or blistering of the skin, and quite as rapid as that previously observed in simple alcohol-prepared incisions.

Pylephlebitis Complicating Appendicitis.—BABLER (*Ann. Surg.*, 1915, lxi, 589) says that formerly it was customary to regard perforative peritonitis as the most frequent and grave complication of appendicitis, but with the almost practical elimination of perforative peritonitis, he believes that the most grave complication is pylephlebitis with multiple abscesses of the lung or of the liver. In any given case the diagnosis must rest on (1) the history showing that the appendix was the primary seat of trouble, (2) the shifting of the symptoms from the appendix to the hepatic region, (3) the progressive increase in the severity and character of the symptoms, (4) the repeated chills followed by high pulse rate and marked elevation of body temperature, (5) the jaundice, (6) the persistent pain in the hepatic region, (7) the urinary and blood findings, (8) the change of liver dulness, (9) the picture of marked toxemia, and (10) the absence of the signs and manifestations of extensive peritonitis. The prognosis is necessarily grave. Prior to the work of Körte and Franke the complication was regarded as practically always fatal. The prognosis depends on (1) the size, multiplicity, and location of the abscesses, as well as their accessibility from a surgical stand-point, (2) the time of their dis-

covery and (3) the general condition of the patient together with the character and time of the treatment. The treatment is outlined as follows: The appendix should be removed, the infected lymphatics leading from the appendix to the liver should be incised and drained, and the liver should be exposed and the abscesses located and drained. The abscess or abscesses in the lung must be located and drained. The intense pain will frequently guide the surgeon to the site of the abscesses. Babler does not feel that it is advisable to aspirate the liver until the latter has been duly exposed, unless preparations for immediate intervention have been completed. He strongly supports the lumbar drain in these cases.

The "Tongue-Depressor" Gastro-enterostomy Clamp. — GIBSON (*Ann. Surg.*, 1915, lxi, 604) illustrates a very simple clamp for gastric and intestinal anastomoses, consisting of three small pieces of wood, the ordinary wooden tongue-depressors, which are bound together by any suitable method, such as strong artery clamp, or, if more convenient, a sterile rubber band. These might be replaced by strips of cigar boxes. In his hands, both in experimental and clinical work, it has proven much more satisfactory than the Roosevelt, Bartlett, and other clamps used for this purpose. He has found it particularly satisfactory in doing a gastro-enterostomy, when it is desired to release the clamp in putting in the last mucous suture and the final peritoneal suture. Removing the rubber band allowed the tongue depressors to fall apart without further manipulation. By this method injury to the viscera is less likely to be produced. Two gastro-enterostomies and one entero-anastomosis have been successfully done with the aid of these clamps.

The Bone Graft Peg in the Treatment of Fractures of the Neck of the Femur.—ALBEE (*Ann. Surg.*, 1915, lxii, 85) says a large number of these fractures occur in individuals below the age of forty-five and fifty. The terms intracapsular and extracapsular are inaccurate and misleading and there is no object in classifying these fractures further than by the single term, "fractures of the neck." If any classification is used that of Stimson is by all means the best, *i. e.*, subcapital, or fracture through the neck, and fracture at the base of the neck. A strong autogenous bone peg, accurately fitted into a hole drilled longitudinally through the neck of the femur, with the fragments in good position, offers unquestionably the most ideal condition for the rapid and satisfactory union, in good position, of this fracture. The technic of the operation is as follows: The patient should be placed upon some traction table (Hawley) which will allow, simultaneously, abduction and traction. The point of the fracture is reached by an incision starting from a finger's breadth inside of the anterior superior spine and curved downward three to five inches along the inner border of the sartorius, which is retracted outward. The rectus femoris is retracted outward and the iliopsoas inward. All soft tissue is then cleared from between the fractured ends, which are curetted and freshened. The limb is now abducted and sufficient traction applied to bring the fragments into good apposition as determined by both sight and touch through the anterior wound. An incision two

to three inches long is then made over and just beyond the great trochanter which is exposed. With a small hand drill the proper direction for the motor drill is determined by trial, as shown by observation through both wounds, the hole passing parallel to and through the centre of both fragments. The motor drill should be held ready for insertion into the tract of the hand drill as it is withdrawn by the assistant. The motor drill, which forms a hole three-eighths of an inch in diameter, is pushed through the distal fragment until the burr end of the drill appears between the fragments, as seen through the anterior wound. Just as the end of the drill is engaging the broken end of the proximal surface, a reading on the graduated drill shaft is taken at its entrance aperture in the trochanter, so that by making additional readings it can be determined how deep the capital fragment is being penetrated. By studying the roentgenogram, the length of this fragment can be very accurately determined, and hence the desired depth of the drill-hole obtained. When the fracture has occurred near the head and the proxinal fragment is consequently short, the drill-hole should extend close to the articular cartilage of the head. The drill is disengaged from the motor and left in place, to avoid any possible displacement of the fragments while the tibial graft is being procured. The crest of the lower portion of the tibia is laid bare, and an area of the desired shape and size is mapped out in the periosteum with a scalpel. The desired length of graft can be determined by the graduated scale on the motor drill. The cross section of the graft should be just large enough to be shaped into the peg when the dowel shaper is used. When the graft peg is ready, the drill is withdrawn from the femur and the peg inserted. The fit must be accurate because the dowel cutter is the counterpart of the drill used. The accuracy of fit is very important. Too tight a fit causes a pressure anemia of the surrounding cancellous bone. Too loose a fit, or an irregular, inaccurate fit, would not produce good fixation or favor an immediate bony union of graft to the host fragments. The deep fasciæ are approximated with interrupted sutures of No. 2 chromic catgut. The skin wound is closed with continuous suture of No. 1 chromic catgut. The limb is placed in abduction (Whitman) in a plaster-of-Paris spica extending from the toes to the axilla. Three weeks after the operation, windows are cut in the plaster, and the wounds dressed. The dressing should be replaced with cotton for the purpose of restoring the tension of the cast and retaining the fixation. The long spica should be continued for six weeks and followed by a short one for six weeks longer.

Cryptogenetic Peritonitis with Special Reference to the Mode of Infection of the Peritoneum.—BRUNZEL (*Deutsch. Ztschr. f. Chir.*, 1915, cxxiii, 233) says that an advance was made with the discovery of the Fränkel-Weichselbaum pneumococcus and the designation of these so-called spontaneous or idiopathic cases of peritonitis according to this, apparently, most common cause. But the new name, "pneumococcus-peritonitis," did not throw light on the mode of infection of the peritoneum. Other bacteria may be at fault as the streptococci and staphylococci. The term, cryptogenetic, is used here to designate those cases of peritonitis in which the cause is unknown or not definitely

established. The most frequent cause found in the cases studied by Brunzel was the pneumococcus. He advises early operation, since the diagnosis cannot be made beforehand with certainty, although the prognosis is not good even with operation. The infection of the peritoneum occurs exclusively by the blood, in all probability. Therefore, the so-called idiopathic or cryptogenetic cases of peritonitis are more properly cases of metastatic peritonitis and the metastases should be regarded as suppurative from a general septic blood infection. The abdominal symptoms occurring in the beginning or during the course of a pneumonia may be regarded as the result of a peritoneal reaction to the flooding of the peritoneum with pneumococci. When the infection gains the upper hand of the peritoneal resistance, the peritonismus becomes a peritonitis. The cases reported as localized pneumococcus peritonitis are to be strictly differentiated from those of general peritonitis. The prognosis is more favorable and for the greatest part, at least, these cases are confused with abscesses of the space of Retzius, which may be metastatic by the blood path or the result of the breaking down and extension of the suppuration from an infected lymph node.

Neurolysis of the Brachial Plexus.—MEHLER (*Deutsch. Ztschr. f. Chir.*, 1915, cxxxiii, 299) reports from his experience in the present European war, the following case in which a gun-shot wound is supposed to have passed through Erb's point in the brachial plexus (junction of fifth and sixth roots of the plexus), and was followed by a complete flaccid paralysis of the arm which is supposed to have been due to the rupture of the plexus at Erb's point. The soldier was wounded October 2, 1914, and admitted to the hospital October 9, 1914. The wound of entrance was at the upper inner angle of the right scapula, the wound of exit just above the clavicle at Erb's point. The right arm was completely motionless (flaccid paralysis). Sensation was preserved. On examination the right arm hung flaccid, active movements were impossible, slight finger movements were present and all reflexes were lost. There was no response to the galvanic or faradic current, from the brachial plexus or the individual nerves and muscles. The condition remained unchanged to the end of December. The region of the brachial plexus was exposed by operation, January 5, 1915. Above and below the clavicle, the plexus was widely and firmly adherent to the surrounding tissues and extended as a broad plate, indistinguishable from the surrounding tissue, and showed tense cords especially posteriorly and lateralward. The plexus was separated, in part bluntly, in part by dissection, and the wound closed. Primary healing followed. Examination on January 22, 1915, showed a weak response of the plexus to the faradic and galvanic currents. There was slight movement of the arm, beginning supination and improved finger movements. On February 13, 1915, the hand and finger movements were almost normal and in the elbow-joint there were slight flexion and extension. The deltoid showed mild contraction. Still further improvement was noted on March 24, 1915.

THERAPEUTICS

UNDER THE CHARGE OF

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The Medical Treatment of Peptic Ulcer with Especial Reference to the Lenhartz Treatment.—BLUMER (*Johns Hopkins Hosp. Bull.*, 1915, xxvi, 127) discusses the subject of the medical treatment of gastric ulcer, referring especially to the various diets as advocated by Leube, Lenhartz, Strauss, Jarotsky, and others. He believes that most patients with acute peptic ulcers, those with hemorrhage of the fulminating type and those with uncomplicated chronic ulcers should have the benefit of a carefully regulated and supervised medical regimen before being submitted to the knife. Ulcer patients with acute perforation, those with pyloric obstruction even in the early stages, those suffering from the results of repeated small hemorrhages intractable to medical treatment, and those with subphrenic abscess, hourglass stomach, or serious perigastric adhesions are best treated surgically. Blumer says that the contrast between different methods of medical treatment lies essentially in differences in the diet. A strict classification upon dietetic grounds is not entirely possible but three fairly sharply defined groups of diets can, however, be recognized; one in which more or less complete abstention from mouth feeding is the predominant feature, typified by the Leube cure; one in which immediate feeding with albuminous food is the essential peculiarity, typified by the Lenhartz diet; and one in which fats constitute the important source of nourishment, typified by the diets of Strauss and Jarotsky. The essential features of the Leube treatment are: Rest in bed; the daily use of luke-warm Carlsbad water; the application of hot poultices and Preissnitz compresses to the epigastrium; and a diet mainly of milk in increasing quantities, fortified after ten days by the addition of cereals and finally by meat. In bleeding ulcers the diet is preceded by several days of starvation, with nutrient enemata, an ice-bag to the epigastrium, and adrenalin, bismuth, and morphin medication. The principle underlying this diet is that to encourage healing of the ulcer the stomach must be spared from work as much as possible, and that in bleeding ulcers the organ must be given complete rest for a time. Modifications of the Leube diet have taken two opposite directions. On the one hand, starvation as a means of resting the stomach is carried to extremes by Rolleston, Wynter, and others of the English school of clinicians. On the other hand, diets advocated by Einhorn, Albe, and Boas increase the caloric value of the Leube diet by the addition to it of easily digestible and non-irritating nutriments. The essential features of the Lenhartz regimen consists of absolute rest in bed for at least three weeks, an ice-bag to the epigastrium until no occult blood appears in the feces, the routine adminis-

tration of bismuth in all patients and of soft Blaud pills in anemic patients, and a non-irritating and non-stimulating diet of concentrated albuminous food given by the mouth from the onset of treatment and gradually increased in caloric value. The principles underlying this diet are: First, the hyperacidity plays an important role in preventing healing and that free acid must be neutralized by acid-binding food; and second, that the general nutrition must be maintained and anemia must be combated to favor healing of the ulcer. This diet has been frequently modified but only in non-essential details. Two diets which have apparently not been widely used, and which are based on the high caloric value of fats and their power to inhibit gastric secretion, are those of Jarotsky and Strauss. Jarotsky's diet consists of white of egg and olive oil given separately, several hours apart, in increasing daily amounts. Strauss uses a mixture of cream and yolk of egg either beaten up with sugar or rubbed up with butter. Blumer notes various objections to these diets as raised by different observers. The one great objection to the fat cures (Strauss, Jarotsky) is the inability of most patients to tolerate the large quantities of fat which they contain. The objections to both the Leube and Lenhartz treatment have been numerous. The best answer to the objections to the Leube treatment lies in Leube's record of 627 patients with a total mortality of 0.3 per cent., and a mortality in bleeding cases of 2.5 per cent. Only 1 per cent. of his patients showed no improvement under treatment, and 90 per cent. showed complete relief of symptoms on discharge. Lenhartz's 295 patients were all subjects of bleeding ulcer, and both his subjective cures and his mortality (2.3 per cent.) are almost identical with the results of Leube. Fewer recurrences of the bleeding during treatment seem to occur with the Lenhartz method than with the Leube. Blumer notes his personal experience with the Lenhartz diet in 27 cases treated with unessential modifications of the diet. In general he found that the diet was, as a rule, eminently satisfactory from the patient's point of view. The pain usually disappears within a few days. The patients seldom complain of hunger, and although most patients lose weight during the first week or ten days, many more than regain this initial loss before the treatment is ended. Thirteen of the patients reported by Blumer were suffering from the symptoms of duodenal rather than gastric ulcer, and 10 of these were entirely free from symptoms or signs of the disease at the end of treatment. All were benefited, there being no absolute failures. Five of them have remained well for over a year and a half. Of 14 gastric cases, two were not benefited at all, and four relapsed after a comparatively short interval. Blumer believes that patients with gastric ulcer do less well on the Lenhartz diet than those with duodenal ulcers. He is of the opinion that ulcers near the pylorus tend to heal badly, while bleeding ulcers seem to heal unusually well. Many ulcers would doubtless heal on complete rest, plus any of the dietary regimens that have been discussed. The tendency to adhere too closely to a particular form of diet is to be condemned as there are good points in all of the diets. The different regimens should be modified or combined in such a way as to suit the needs of the individual patient. The author gives the details of the various methods of dietetic treatment in his article.

Artificial Pneumothorax.—LEUT (*Jour. Amer. Med. Assn.*, 1915, lxiv, 1973) sums up the indications for and the contraindications against the induction of an artificial pneumothorax for the treatment of pulmonary tuberculosis. He says that the ideal case is one in which the disease is limited to the upper portion of one lung, an acute progressive condition, generally with signs of softening, but which has not responded to the usual therapeutic measures. The treatment is indicated in cases with marked involvement of one lung, with a moderate infiltration of the opposite lung, preferably the apex. Artificial pneumothorax has often given brilliant results in cases of severe and uncontrollable hemorrhage. Some cases of pulmonary tuberculosis complicated by pleurisy with effusion, offer an excellent opportunity for giving compression treatment. Lung abscesses and bronchiectasis are occasionally benefited. Complications, such as tuberculous laryngitis or enteritis, may improve if the patient is doing well otherwise. The chief contra-indications are as follows: An extensive or progressive lesion in the opposite lung, or a disseminated miliary tuberculosis would preclude the gas treatment. The treatment would not be justifiable in patients even though far advanced who are doing well under the usual therapeutic measures. Generally, basal lesions in the opposite lung are contra-indications and are less favorable in the collapsed lung. Endocarditis and nephritis would lessen the chances of recovery in proportion to their severity. While dense pleural adhesions could not, strictly speaking, be considered contra-indications, they are nevertheless the chief cause of a large percentage of failures. Lent notes some of the complications that may follow the induction of artificial pneumothorax. Pleurisy with diffusion may occur in as high as 50 per cent. of the cases. This complication, though rarely serious, is an annoying one. The fluid persists for a long time, but generally should not be removed unless causing serious symptoms. The fluid may then be withdrawn and nitrogen gas introduced in its place. Tuberculous pyopneumothorax occurs in a small percentage of these effusions, but does not necessarily give a bad prognosis under the expectant treatment. Some cases of secondary infections have been reported, in which cases the outlook must be regarded as very serious. Numerous cases of shock following the operation have been reported. This can probably be avoided by careful cocainization of the tissues and by a preliminary injection of morphin. The danger of gas embolism is remote with proper technique. Emphysema, superficial or deep, may be encountered, but rarely produces any serious symptoms. Puncture of the lung followed by slight bleeding, may occur, but generally does not give rise to any serious symptoms. Dilatation of the heart is a serious complication, and is usually due to too great intrathoracic pressure. Care in gradually inducing the pneumothorax to permit the heart to accommodate itself to the increased pleural pressure should obviate this possibility. More or less severe pain caused by breaking up of pleural adhesions may also be mentioned among the complications. In his conclusions, Leut states that it is most gratifying that at last we have a rational treatment that may be used in the far-advanced cases. A comparatively few years ago symptomatic treatment, with the hope of temporary relief, was the only one given. The present status of artificial pneumothorax is that in a fair percentage of these

hopeless cases we can offer either an arrest of the disease, or a chance of returning to good health. The procedure is attended with slight dangers, but these are insignificant as contrasted with the fatal outcome if the disease is allowed to go on unchecked. The duration of treatment is a question offering the greatest problem. It is impossible to determine whether the tuberculous foci in the collapsed lung have become entirely cicatrized, even though the patient is clinically well. It is far better to continue the treatment for an indefinite period, extending over years, than to discontinue the compression and discover after a few months that the disease is becoming active again, and when an attempt is made to readminister gas, that the layers of the pleura are densely adherent.

The Treatment of Leukemia. — HAHN (*Therap. Monatsh.*, 1914, xxviii, 555) says that during the past few years there has been a greatly increased literature dealing with the treatment of leukemia and that considerable discrepancy of opinion exists among different observers. From a study of the literature and his own observations the author gives his views regarding the present status of the treatment of leukemia. He believes that leukemia should be vigorously treated only where there is a severe and progressing anemia, the presence of large leukemia tumors or a great increase of the white cells. It is now recognized that many cases of leukemia suddenly show a marked aggravation of the disease even when apparently doing very well under some form of treatment. The relapse in such a case may prove far worse than the disease originally was when treatment was begun. With regard to the different methods of treatment, the author states that the Roentgen-rays seem to check the activity of the tissues producing white blood cells, but overdosage may prove fatal, and too small doses may actually stimulate the production of white cells. The stimulating effects of Roentgen-rays on the production of white cells have been observed in professional roentgenologists; Kimecke has recently reported six cases of leukemia developing in Roentgen-ray workers as a result of long-continued exposure to the rays. Hahn advises systematic exposure of every accessible gland in lymphatic leukemia, but in myeloid leukemia exposure of the enlarged spleen alone is effectual. In cases resistant to the treatment, benefit may be obtained by exposing the long bones of the legs between series of other exposures. He notes a marked improvement in 75 per cent. of his cases, while the others were not influenced. Béclère has reported no failures in 12 lymphatic and 93 myeloid cases and still others report very favorable results. Lymphatic leukemia seems less influenced by roentgenotherapy than myeloid leukemia, and almost invariably relapses. The effect of radio-active substances seems to be very similar to that derived from the Roentgen-rays. Radio-active substances fail in about 20 per cent. of the cases, according to the cases reported in the literature. Hahn found a great difference of opinion regarding beneficial effects of benzol therapy. He says it should not be given with coexistent disease of the liver or kidneys or when catarrhal intestinal inflammation exists. The consensus of opinion is that its use should not be continued till the leukocytes fall to normal numbers but that it should be discontinued when the leukocytes have dropped to 20,000 or 25,000. Hahn says

that combined treatment is often very effectual especially a combination of benzol and roentgenotherapy. When combined, both can be used with lesser dosage. His experience has been that better results are obtained when treatment is begun with the Roentgen-rays and continued with benzol, not giving more than one gram a day of the latter. If myeloblasts appear in large numbers under roentgen treatment he changes to thorium X and follows with vigorous treatment with arsenic. In case of flaring up of the leukemia while under roentgenotherapy arsenic should be given a trial. With acute leukemia all treatment seems to be hopeless, but as it is not always possible to differentiate the acute form Hahn advises the trial of thorium X combined with a vigorous course of arsenic.

Experiences of the New York Board of Health in Typhoid Immunization.—HARRIS and OGAN (*Jour. Amer. Med. Assn.*, 1915, lxiv, 3) say that accurate observations recorded in hundreds of thousands of cases leave no doubt as to the preventative powers of antityphoid vaccination in all but a relatively insignificant number. In those few cases subsequently affected, vaccination strikingly decreases the morbidity and the mortality. Severe reactions are rare, but in order to avoid such reactions it is necessary to carefully observe certain precautions. Antityphoid vaccine should never be administered to any but the healthy. In order to permit of slow absorption the puncture of a vein, or intramuscular injection must be avoided. Children especially should avoid exposure to the sun following the administration of the vaccine. Antityphoid vaccine should not be given during the menstrual period or during pregnancy. No hard work or indulgence in alcohol should be allowed after the injection. Care should be taken not to reinject the vaccine in indurated areas. The authors state that when the incubation period has begun, the time for antityphoid vaccination has passed. Long and continued exposure to overwhelming doses of typhoid bacilli (in those who are in close contact with cases and especially in epidemics) may nullify the immunizing process of antityphoid vaccine, and an attack may therefore follow one or more injections. In fact antityphoid inoculation seemed to hasten the onset in cases cited by the authors. Chronic illness such as tuberculosis as well as debility or fatigue from other causes predisposes to severe reactions. The disease may develop after a complete immunizing course of treatment, in exceptional instances in which debility and fatigue exhaust the defensive powers of the body, and when exposure to massive doses of typhoid bacilli exists.

The Treatment of Typhoid Bacilli Carriers.—GEROME and LENZ (*Berl. klin. Wchnschr.*, 1915, lii, 341) were able to render the stools free of typhoid bacilli in three men, who were apparently becoming chronic carriers by giving thymol in combination with animal charcoal. Thymol was selected as an efficient disinfectant and the charcoal was combined with it on the theory that the charcoal would absorb the thymol and thus inhibit its absorption by the tissues and permit it to act as an intestinal antiseptic for a longer period of time and over a greater portion of the intestinal tract. This theory seemed to be confirmed by the urinary examinations which denoted much slower ab-

sorption of the drug. The thymol and charcoal were given in doses of one gram each three times a day; the charcoal half an hour before meals and the thymol half an hour after meals. These doses were continued for from one to two weeks. The bacterial content of the stools of normal individuals was tested after charcoal alone and after thymol alone and showed a distinct reduction but the diminution in bacteria was far greater when they were given together. The authors are of the opinion that this method is an efficient way of inducing intestinal disinfection.

OBSTETRICS

UNDER THE CHARGE OF

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The Disinfection of the Skin Before Operation.—At a Meeting of the Obstetrical Society of Berlin, FRANZ (*Ztschr. f. Geburtsh. u. Gynäk.*, 1915, lxxvi) stated that the use of the sterile rubber glove had solved the problem of disinfecting the hands. The question still remains as to what should be done with the skin at the field of operation. We may recall the elaborate methods which have been tried, consuming from twenty minutes to a half hour. The use of iodine has made such disinfection much shorter and less complicated. Franz compares the results obtained by the use of iodine in disinfecting the skin at the field of operation during one year. There were 273 abdominal sections, and as some were Alexander operations with double incisions there were 334 incisions. The mortality was 5.4 per cent. Perfect healing without secretion occurred in 81.7 per cent. Wound abscess formed in 6.9 per cent., and there was secretion in the wound in 6.6 per cent.

He compares these results with the work of another year, in which the following method was used: On the day preceding operation, the patient had a very thorough general bath. In some cases the abdomen was cleansed with cotton dipped in alcohol. No other local disinfection was practised. In this series there were 324 abdominal sections, with 81 Alexander operations, the latter making 405 incisions. The mortality was 4.6 per cent., while 85.9 per cent. of the cases had perfect healing without complications. Abscess formed in the wound in 8.1 per cent., and there was secretion in the wound in 3.3 per cent. When the two series are compared the results are all in favor of the bath and occasional alcohol method only, except in the occurrence of abscess in the wound. By the iodine method there was a smaller percentage than by the other, but this is counterbalanced by the fact that in the simpler method secretion occurred much more rarely in the wound. In discussion, Fromme described experiments which he had made upon some of the pregnant smaller animals to obtain their young in absolutely sterile conditions. To do this the young were delivered by Cesarean section, in a carefully sterilized room, and were then fed upon sterile food to avoid any possible con-

tamination with bacteria. The young so delivered were stronger and grew much faster than those born in the ordinary way. The room was so constructed that manipulation could be performed with the hands covered with rubber gloves, without admitting air which contained bacteria. The further experiment was tried of making abdominal incisions upon these sterile animals to determine the healing of the wound. When the stitches were removed the wounds were found perfectly clean, but the edges were not in immediate apposition. The result would indicate that some bacteria are a benefit to the healing of the wound. Jolly recalled Olshausen's habit in cases of recently-ruptured, ectopic pregnancy, to operate without preparing the abdominal wall at all, and simply pour alcohol over the instruments. The results were excellent. No patient died of septic infection, and the wounds healed without complications. Jolly believed that the skin at the umbilicus was most dangerous through retained secretions and bacteria. Hammerschlag had tried applying wet dressings of bichloride solution to the abdominal wall for twelve hours preceding operation. He had seen one fatal bichloride poisoning as a result. Bumm had noticed that with the same precautions a series of cases would proceed with excellent healing of abdominal wounds, and then would come a series where stitch abscess and other complications would arise. At times in a Clinic nurses and physicians show a tendency to suppurating eruptions of the skin. Bumm described this to be the gradual development of bacteria in the hospital itself, and especially where cases were brought in infected, or where malignant growths proceeded to suppuration.

Abdominal Cesarean Section Under Local Anesthesia.—WEBSTER (*Surg., Gynec., and Obst.*, May, 1915) adds three others to his list of cases of Cesarean section under local anesthesia. The first patient had been in labor twenty-four hours and was somewhat exhausted, the fetal head very hard, large and above the pelvic brim, the cervix dilated. The operation was successful, the child weighing nine pounds, ten ounces. The patient remained quiet throughout the operation. The second patient was a girl of thirteen years and some months at full term pregnancy, the head to large to pass the pelvis. The child weighed eight pounds, three ounces, and the mother remained quiet during the operation. The third patient had placenta previa and pulmonary tuberculosis. Novocain was used with her, and to prevent further impregnation, the Fallopian tubes were divided. The results were satisfactory. He also reports the case of a woman who had a hernia of the fundus uteri. The hernial sac which was composed of the fundus was about three inches wide, on the left of the middle line, rising about an inch and a half above the level of the uterine wall. When the uterus contracted the hernia was made more prominent by the amniotic liquid forced into it. On section the hernial portion was removed, as it was only one-eighth of an inch thick. He also performed section under local anesthesia upon a woman, who, in a previous pregnancy had fallen on a bread knife which had perforated the anterior abdominal wall and uterus, necessitating an abdominal Cesarean section. Within two months of this operation she again became pregnant. At the second operation, the uterine incision was

made two inches from the old scar and parallel with it. The old incision was found very thin and the tissue was dissected out to secure good union. To prevent the pain of infiltration for local anesthesia caused by the introduction of the needle, Webster has given nitrous oxide gas. In this way infiltration can be performed without haste and large areas of the skin rendered without sensation.

Anesthesia in Labor by Nitrous Oxide Gas.—Webster (*Surg., Gynec., and Obst.*, May, 1915) describes the use of nitrous oxide gas alone, or mixed with oxygen, 3 per cent. With the use of a small inhaler that covers the nose alone during the pain of contraction, sufficient gas may be given to abolish consciousness without lessening the pains. This method is used during the second stage, the anesthetic is withdrawn between the pains. The expense need not exceed $15 for any case, and may be reduced to about $5, with care.

Nephrectomy During Pregnancy.—Harrigan (*Surg., Gynec., and Obst.*, June, 1915) describes the case of a patient, four months advanced in her first pregnancy, who had an alveolar abscess. This ruptured spontaneously, but the patient developed a chill with high fever. Examination of the blood revealed a very high leukocytosis and the symptoms pointed to unilateral hematogenous infection of the kidney. Accordingly the right kidney was removed and found to be the site of multiple suppurative infection. The patient went on in pregnancy until seven and one-half months, when an attack of toxemia developed, and labor was induced and a viable child was born, which survived. Two years after the patient was in excellent health and apparently suffered no inconvenience from the loss of the kidney. Harrigan has collected 36 cases from the literature, and among these the ultimate result is stated in 30, of whom 28 recovered. Of these 20 went to labor without accidents or complications. Two aborted, and in two abortion was induced. Should the indications be present the obstetrician need not hesitate to remove the kidney from a pregnant patient. The reviewer has seen several women having but one kidney pass through pregnancy and labor successfully. In one instance a woman whose kidney had been removed for tuberculous infection and was without adequate care during pregnancy, had eclampsia from which she recovered.

The Duration of Pregnancy.—Peters (*Zentralb. f. Gynäk.* No. 17, 1915) discussed this subject before the Obstetrical Society of Vienna. He calls attention to methods already in use for estimating the duration of pregnancy, which fix the average at 270 days. In comparative studies with cows, in over 3000 cattle, the average was 284 days, with a variation between the longest and shortest pregnancies of 72 days. He quotes observations in Winckel's Clinic, where in 16,000 labors pregnancy in 31 cases had lasted from 302 to 322 days; in 12 cases 291 days. In the general series, in 71 per cent. pregnancy had lasted more than 280 days after the termination of menstruation, and in 62 per cent. pregnancy lasted over 270 days after conception. In general from 2 to 3 per cent. of pregnancies were considerably prolonged. The weight of the child increased in proportion to the length of pregnancy. As an aid in estimating the duration of pregnancy it must be remembered that ovulation occurs from fifteen to nineteen days

after the beginning of the last period and from thirteen to nine days before the beginning of the next period. The average period of pregnancy after conception must be taken as practically 270 days. If we calculate more closely upon these data the average duration of pregnancy is from 261 to 268 days. It must be remembered that variations in the nutrition of the fetus play an important part in the duration of pregnancy, also variations in the type of menstruation in the mother. The influence of season, climate, race, and many other incidental factors affect this problem. In discussion, Schauta believed that the best method of practical reckoning was to consider pregnancy as enduring 270 days after conception and 280 days from the last period. He draws attention to the many difficulties in exact computation. He would take the first day of the last period, record the day of labor, and from this sum total of days subtract 18 or 19. This would give the probable time of pregnancy.

The Effects Produced by Placental Extract.—Colle (*Ann. di Ostet.* No. 4, 1915) has experimented with placental extract by injection into animals. He finds that when placed directly in the circulation it causes toxic symptoms, that when introduced into other channels of the body, this toxic element is absent. When injected into the veins it causes increased coagulation. It produces a lessening of blood-pressure of variable duration without excitement of the vasomotor centre. It has a tendency to lessen the respiratory rate and has little influence upon the heat centre. In a normal pregnant patient it does not bring on premature labor, but may stimulate the occurrence of labor at term. The maternal portion of the placenta is less active in its results than the fetal portion. The blood obtained from the placenta has the same effect as placental extract. When placental extract is made before the placenta has been thoroughly washed of all soluble material the extract is more efficient than if the placenta had been cleansed of its products. The efficiency of placental extract is in inverse proportion to its concentration. Putrefactive changes in the placenta diminish the efficiency of its action. The nitrogenous contents of the placenta vary in different cases and with different species of animals. Experiments show that the effect produced by placental extract may vary greatly in different cases. The effect upon an animal varies considerably with the rapidity of injection, for the rapid introduction of a large quantity of placental extract may prove fatal. In these cases no evidence of thrombosis or embolism is found. When placental extract is subjected to the action of the centrifuge there seems to be no essential difference in the material obtained.

The Delivery of the Placenta.—Kirkpatrick (*Jour. Obst. and Gynec. Brit. Emp.*, January, 1915) writes concerning methods employed to secure the delivery of the placenta. He finds that in 1767 Harvie published a pamphlet in which he called attention to the danger of pulling upon the cord, and recommended pressing downward and toward the pubes upon the uterus to expel the placenta. This method was not the Credé method, in that it did not compress the uterus from before backward, but it was a great improvement upon the delivery of the placenta by pulling upon the cord, and foreshadowed the Credé method.

HYGIENE AND PUBLIC HEALTH

UNDER THE CHARGE OF

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The Schick Reaction.—SCHICK, of Vienna (*Münch. med. Wchnschr.*, November 25, 1913, 2608), and PARK, of New York (Park, Zingher, and Serota, *Arch. Pediat.*, 1915, xxxi, 481) find that the blood serum of about 80 per cent. of the newborn, from 50 to 60 per cent. of children, and 90 per cent. of adults contains sufficient diphtheria antitoxin to protect them against diphtheria. A positive Schick reaction is sufficiently delicate to indicate less than $\frac{1}{30}$ of a unit of antitoxin in 1 c.c. of blood serum. Such persons are susceptible to diphtheria. The Schick reaction consists in injecting $\frac{1}{50}$ of a minimum lethal dose of diphtheria toxin for a 250-gram guinea-pig into the skin. BUNDESEN (*Jour. Amer. Med. Assn.*, 1915, lxiv, 1203) concludes as follows: (1) By means of the Schick test we are in a position to tell definitely who is susceptible to diphtheria and when an epidemic breaks out can inject those and those only, paying no further attention to the one giving a negative reaction. (2) By it the danger of cross-infection is greatly decreased. Children with diphtheria that have had scarlet fever and are therefore immune from scarlet fever can be placed in scarlet fever wards when the inmates yield negative Schicks, etc. (3) It permits a great reduction in antitoxin bills. Much needless pain and annoyance of patients is avoided and the possibility of anaphylactic shock is greatly minimized. GRAEF and GINSBERG (*Jour. Amer. Med. Assn.*, 1915, lxiv, 1205) conclude as follows: (1) The most susceptible age is between one and five years. (2) Immunity obtained by having the disease or by the use of immunizing doses of antitoxin lasts from a month to several years, varying greatly in different individuals and being very brief in children. (3) The Schick test is a helpful agent in testing the efficiency of immunization by antitoxin as well as the natural immunity existing in many persons. (4) It has helped to place on more certain grounds the assurance that bad cases of diphtheria should receive early and large doses of antitoxin by intravenous injections. Antitoxin is ten times as effective when so used, as compared with the ordinary methods of administration. (5) Park has obtained results in families showing a striking similarity in reactions to the test. If the youngest child of a family has a negative reaction, all the older children are likely to be negative, and if the older children are positive, the young ones are also. When variations are found, the younger children show the positive reaction. MOODY (*Jour. Amer. Med.*

Assn., 1915, lxiv, 1206) concludes as follows: (1) The test is of definite clinical value in differentiating between persons who are susceptible to diphtheria and those who are not susceptible, and is therefore useful in determining which cases, of those to be placed in an environment where the liability to exposure is great, should be immunized. (2) It is of great value in differentiating between clinically doubtful cases of diphtheria, positive reactions being obtained in clinical cases, negative reactions in carriers. (3) It is useful in experimental work in determining the effects of various forms of immunization and the duration of immunity conferred by these methods. (4) It has a definite value in the handling of diphtheria epidemics in institutions. KOLMER and MOSHAGE (*Amer. Jour. Dis. Child.*, March, 1915) studied "The Schick Toxin Reaction for Immunity in Diphtheria," and summarize their observations as follows: (1) The toxin skin reaction is a valuable and reliable method for detecting susceptibility to diphtheria. (2) Persons reacting negatively to this test usually contain at least $\frac{1}{20}$ unit of diphtheria antitoxin per cubic centimeter of serum, and this amount of antitoxin is probably sufficient to protect against infection. (3) Persons reacting weakly or strongly positive usually contain less than $\frac{1}{40}$ of a unit of antitoxin per cubic centimeter of serum or none at all. These persons may be regarded as susceptible to diphtheria and in the event of exposure to infection should be passively immunized with an injection of antitoxin. (4) About 40 or 50 per cent. of children, ranging from one to fifteen years of age, react positively to the toxin test; this means that the preliminary use of the toxin test will eliminate the necessity of administering prophylactic doses of antitoxin to about 50 per cent. of children. (5) The toxin reaction indicates that the immunity conferred by an injection of antitoxin begins to disappear after ten days and has generally passed away entirely after four weeks. (6) The increased susceptibility of persons with scarlet fever to diphtheria is shown by the toxin reaction; even after the injection of antitoxin about 10 per cent. are susceptible within ten days. (7) According to the toxin reaction the immunity conferred by an attack of diphtheria is usually of short duration or entirely absent. (8) The most practical application of the toxin reaction consists in applying the test as a preliminary measure to all persons who have been exposed to diphtheria and immunizing only those who react positively.

Pellagra.—The Thompson-McFadden Pellagra Commission of the New York Post-Graduate Medical School and Hospital has just issued its second progress report, in which it presents data confirming the previous impression that the disease is communicable and in some way associated with insanitary methods of sewerage disposal. These conclusions are directly opposed to those of Goldberger, of the Public Health Service, whose studies led him to believe that pellagra is due to a faulty dietary. The conclusions of the Thompson-McFadden Pellagra Commission, consisting of J. F. Siler, P. E. Garrison and W. J. MacNeal, are as follows: (1) The large active foci of pellagra in Spartansburg County were found in and near the large centres of population, and particularly in the cotton-mill villages. (2) Children under the age of two, adolescents for about five years following puberty, and adult males in the active period of life were least frequently affected by pellagra. On the other hand, women from twenty to forty-four

years of age, old persons of both sexes, and children from two to ten years of age were most frequently affected. (3) No definite connection between occupation and the occurrence of pellagra has been found, although the high pellagra morbidity in the women and children points to the home as the place in which the disease is usually contracted. (4) In the group of incident cases most thoroughly studied, evidence of close association with a preëxisting case was disclosed in more than 80 per cent. (5) A house-to-house canvass of the homes of over 5000 people living in six endemic foci of pellagra failed to disclose any definite relation of the disease to any element of the dietary. (6) In these six villages new cases of pellagra originated almost exclusively in a house in which a preëxisting pellagrin was living, or next door to such a house, suggesting that the disease has spread from old cases as centres. (7) So far as we have observed, pellagra has spread most rapidly in districts where insanitary methods of sewage disposal have been in use. (8) Additional evidence has been obtained to support the conclusion that flies of the genus *Simulium* have nothing to do with pellagra. (9) Animal inoculations and the experimental study of intestinal bacteria have not yielded conclusive results. (10) The studies of the blood have shown a lymphocytosis in most cases, but have not disclosed any constant abnormality characteristic of pellagra. (11) There is no evidence of inheritance of pellagra. (12) The immediate results of hygienic and dietetic treatment in adults have been good, but after returning to former conditions of environment, most of the cases have recurred. In children, prognosis is very much more favorable.

Geographic Distribution of Plague and Its Menace to the United States.—W. C. Rucker (*Public Health Reports*, May 14, 1915, p. 1428), in his article, recites the distribution of bubonic plague throughout the world with special reference to the lines of travel from such localities to the various borders of the United States. The measures to be adopted for the prevention of the introduction of plague into the United States are considered under four general headings. The first of these is the maritime quarantine, which the author states can be absolutely effective only at the cost of great interference with commerce. The second line of defense is at the wharves and consists in the prevention of embarkation or disembarkation of rodents; but this is a measure on which absolute reliance cannot be placed. The third line of defense consists in the immunization of human beings against bubonic plague; but this is at best a poor procedure and, in the absence of an epidemic, is manifestly impracticable. The fourth line of defense consists in rat-proofing the environment in which man works and lives. All other methods of prevention may be used; but the one on which the greatest reliance can be placed, and from which lasting protection may be obtained, is by the insulation of man from the animal which serves as the disseminator of bubonic plague. Any city which will render itself completely rat-proof need have no fear whatever of the disease. The eradication of plague from the city of New Orleans has already cost the Federal, State, and municipal governments upward of $400,000, exclusive of the large sums which corporations and private individuals have expended in rat-proofing. This amount of money would have gone a long way toward building permanent fortifications against rats.

A Microscopic Test For Pasteurized Milk.—Frost (*Jour. Amer. Med. Assn.*, 1915, lxiv, 821) started to work upon the theory that it would be possible by means of an *intra vitam* stain to color live leukocytes. In this way the live cells in raw milk could then be distinguished from the dead cells in heated milk. Frost uses one part of a saturated aqueous solution of methylene blue to five parts of the milk to be tested. The dye should be added to the milk slowly to prevent the possibility of the stain coagulating the milk. The stain is left in contact with the milk for fifteen or twenty minutes, and the sediment of the stained milk is precipitated in a centrifuge and spread on a glass slide. When the smear is dry it is ready for examination. The entire microscopic field is stained a light blue in the case of raw milk. In this blue background appear numerous clear areas, the smaller ones being fat globules, the larger ones leukocytes. On the other hand, the smears made from heated milk do not have the background as deeply stained, and the leukocytes are always more deeply stained than the background. Only the polymorphonuclear leukocytes react in this way, for the mononuclear cells are always well stained.

The Germicidal Action of the Ultraviolet Ray.—Houghton and Lewis (*Amer. Jour. Pub. Health*, 1914, v, 225) conclude that: The ultraviolet rays produced by the Cooper-Hewitt mercury arc have a strong bacterial action. Certain species of bacteria in aqueous suspension, including spore-forming organisms, are killed by exposure to the rays. Molds, however, are only partially destroyed by the ultraviolet light. The action seems to be a photo-mechanical process, and is, in all probability, due to absorption of the ultraviolet rays by the bacterial protoplasm. Water, wines, many inorganic, and a number of organic substances in aqueous solution, can be sterilized by ultraviolet light. Bacterial vaccines require a prolonged action. Proteins and other bodies of high molecular weight interfere with the action of the rays. Turbidity, both organic and inorganic, has a similar action. Color, within certain limits, seems to have no influence.

Protective Inoculation Against Mumps.—A. F. Hess (*Proc. Soc. Exper. Biol. and Med.*, 1915, xii, 144) calls attention to the fact that mumps confers a marked immunity and that second attacks are rare. He therefore used the blood of convalescents as a prophylactic. Six to 8 c.c. was injected intramuscularly in 17 cases. These children were in wards where there had been cases of mumps for the past month and where it continued to appear for a month following these inoculations. In no case did one of the inoculated children develop mumps, whereas one-third to one-half of the non-inoculated cases developed the disease. The blood was taken from children who had just recovered or had been well for about ten days. It would seem that this proceeding could be made use of in the institution as well as in the home, and that this type of therapy could be adapted in the case of measles and other infectious diseases.

PATHOLOGY AND BACTERIOLOGY

UNDER THE CHARGE OF

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Hypertrophy of Right Heart Following Inflammatory Thickening of Pulmonary Arterioles.—SCHUTTE (*Centralb. f. Path.*, 1914, xxv, 483) reports this finding in a woman aged seventy-four years, who died of cardiac insufficiency with generalized edema. The right ventricle was hypertrophied and dilated and formed the apex of the heart. The left heart with its valves was quite normal and the aorta with its branches was unusually free from sclerotic change. Macroscopically nothing was found to account for the hypertrophy and dilatation of the heart. The microscopical study of the lung, however, showed a thickening of the intima of the pulmonary arterioles of certain constant caliber. The vessels of only a particular size appeared with the intimal lesion and the condition was more marked in the left upper lobe than elsewhere. The intimal thickening was of an inflammatory and proliferative kind. No associated lesion was found to explain the localized inflammatory process of the arteries of the lungs.

The Localization of Emboli in the Lung.—Ribbert observed that emboli coming from the inferior vena cava usually localized in the lower lobe of the lung. This was substantiated by Kundrat who found that the emboli of puerperal sepsis passed to the lower lobes while those of otitic origin were found in the upper lobes. The animal experiments of Virchow, however, showed that foreign bodies introduced into the jugular vein of the dog were most commonly found in the lower lobes, while similar experiments by Behring with tubercle bacilli demonstrated the most usual lesion in the upper segments of the lung. Kretz in following human autopsy material came to the same conclusion as Ribbert and his pupil Helly substantiated these results in a series of animal experiments. It would appear from these findings that there are definite currents within the blood mass of the heart and large veins. In fact, Kretz has suggested a theory of these currents by which the blood of the upper cava passes to the upper pulmonary artery and from here goes through the heart in such a manner that it is driven into the aorta and to the lower portion of the body. The blood returning from these parts by the inferior cava is driven to the lower pulmonary system and upon its return to the left heart passes to the aorta so that

it reaches the vessels of the upper extremities and head. These rather far-fetched theories have not been substantiated by others. ALWENS and FRICK (*Frank. Ztschr. f. Path.*, 1914, xv, 315) carried out animal experiments in which they injected bismuth suspensions into the ear or femoral veins. The lungs were examined during life and after death with the Roentgen-ray. By this means they were able to observe the course of the injected materials quite accurately. Besides this, histological examination of the specimens was also undertaken. They were unable to agree with the Kretz findings and could offer no evidence that the blood from the upper and lower portions of the body remained in separate streams. In the living animal they could observe that the blood in the ventricles was fully mixed. They found, however, that the injection of larger quantities of foreign material led to the localization of much of it in the lower lobes of the lung. This localization occurred in this tissue regardless whether it was introduced into the jugular or femoral veins.

Variability of Pathogenic Bacteria.—Bacteriologists have long been interested in the variations developing among pathogenic bacteria. BERNHARDT (*Ztschr. f. Hyg. u. Infectionskrankh.*, 1914, lxxix, 179) has contributed to the subject some important results. He worked chiefly with strains of B. typhosus, and the diphtheria group but also records observations on B. paratyphosus, meningococcus, the bacillus of fowl cholera and the bacillus of rabbit septicemia. The alterations he obtained with the B. typhosus were marked changes in the types of colonies, cultural differences in broth, variations in agglutination, and loss of motility. In his series of experiments he was able to trace all degrees of variations from the original strains to the extreme variants. He also found marked differences in the permanency of these variations. One variant of B. typhosus lost its motility and remained non-motile during the period of observation, one and a quarter years, even after animal passage or frequent transfers. He was unable to find any fundamental differences in agglutination or antigen powers in the altered strains. Most of his variants were obtained from broth cultures at different periods of growth and he found that variants tended to disappear and that types closely resembling the original remained in the older cultures. Among the members of the diphtheria group the principal changes obtained were in morphology, colony formation and toxin production. All graduations in variation were noted. These alterations were mostly from broth, Loeffler's serum and agar, but he also obtained alterations by animal passage. The author believes that the changes obtained upon media also take place in man and supports his view by the finding of atypical forms in the nasal cavity and the urine of diphtheria patients. In two cases, both fatal, he grew true diphtheria bacilli from the urine. Some of his alterations were accompanied by lowered resistance and in others by increased viability. The latter was especially observed in the altered types of the bacillus of rabbit septicemia and meningococcus. The author discusses very fully the theoretical and practical significance of his findings and those of others. He believes that the source of these alterations is to be found chiefly in the metabolic products of the bacteria and that different bacteria react differently to the various stimuli. Moreover, he does not

agree with the view that the bacteria are predisposed by these means and that sudden changes take place. It is a gradual alteration. He also shows that the variations are not necessarily of any benefit to the bacteria but take place irregularly and almost by chance. An attempt is made to correlate these findings with the general problem of heredity and he discusses the use of such terms as mutation, modification and variety and points out that they can only be used in a relative sense.

The Widespread Distribution of Diphtheroids and Their Occurrence in Various Lesions of Human Tissues.—Considerable confusion has been brought to the minds of many investigators by the finding of diphtheroid organisms in a great variety of human tissues. At times, an important relation is suggested between the organism and the diseased tissue; as in Hodgkin's disease and leprosy. Much difficulty is also experienced in designating the limits of the group. In one direction they advance closely to the true diphtheria bacillus, while in the other, transition forms closely approach the streptothrix group. HARRIS and WADE (*Jour. Exper. Med.*, 1915, xxi, 493) have pointed out the wide distribution of this group of organisms in the human body. Under normal conditions it has been shown to be present in the eye, brain and cerebrospinal fluid and blood cultures. The authors have obtained them from lymph glands in simple hyperplasia while others have laid much stress upon the importance of finding the diphteroids in the lymphatic system. Moreover, similar organisms were obtained from benign and malignant tumors of various kinds, but in no instance could any etiological importance be laid to them. Diphtheroid forms are also not uncommon upon the skin and in the air. The various diphtheroid organisms which have been isolated may to a certain extent be differentiated but up to the present no accurate precedure has been developed, whereby a suitable classification may be employed.

On the Mechanism of Pneumococcus Immunity.—BOEHNCKE and MOURIZ-RIESGO (*Ztschr. f. Hyg. u. Infectionskrankh.*, 1915, lxxix, 355) believe that the promotion of phagocytosis is a more important function of pneumococcus sera than the antitoxic activity. From the experiments these authors, along with other investigators, have shown that pneumococcus sera possess a very definite antitoxic principle but that the animals are not cured by its use. The time of death is, however, definitely delayed. These sera promote phagocytosis and by this means exhibit their most important function in curing cases of pneumococcus infection.

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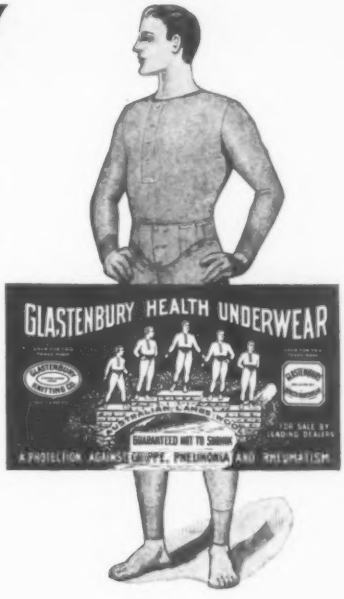

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